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OEDEMA ON MANIHOT

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(WITH PLATE XI AND ONE TEXT FIGURE)

The blister-like or pustular outgrowths on plants, variously designated as oedemata or intumescences, have from time to time been the subject of special investigation. This abnormal or diseased condition, which occurs quite frequently upon the leaves of various plants, may also appear on stems or trunks and has been noted rarely on the flowers and fruits. It has been observed on plants grown both out-of-doors and under glass.

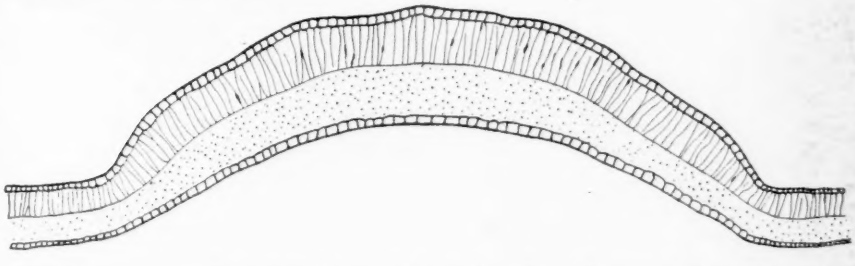

FIGURE 1. A diagrammatic vertical section through one of the blister-like elevations on ceará.

Our attention has recently been directed to a diseased condition of certain rubber-producing plants, *Manihot glaziovii*, *M. heptaphylla* and *M. piauhyensis*, commonly known as "ceará," growing in the greenhouse at the Agricultural Experiment Station, Auburn, Alabama. The surface of the leaves is partially occupied by numerous, glistening, prominently-projecting elevations (Fig. 1). These may occur on either surface of the leaf, although, on any particular leaf, all extend in the same direction so far as observed. When the elevations occur on the upper surface there are corresponding depressions or concavities on the lower. The diseased areas are very small at first

but they may become as much as three millimeters in diameter and protrude a millimeter above the surface of the normal leaf tissue. They are circular in outline and are for the most part isolated. When there are from 300 to 500 of these blister-like processes on a single leaf they may become confluent and consequently more or less irregular in outline. They seem always to appear between the principal veins of the leaf and never to involve them. In the earlier stages of development the intumescences appear as small swellings unaccompanied by any change in color from the normal leaf-green. As the diseased condition progresses, however, the entire outgrowth, beginning at the center, becomes brown and finally the tissues dry and collapse.

Sections through the affected areas show that no fungus is present, and further study reveals no reason for a causal connection with any organism. The cells of the diseased tissues undergo the changes shown in the accompanying illustrations (Plate XI), all of which are drawn at the same magnification with the aid of a camera lucida. There has been a very considerable enlargement and distortion of the cells. The normal arrangement of the leaf tissues is shown in Figure 1 while Figure 4 represents an early stage in the development of the disease in which all the cells, except those of the lower epidermis, are swollen and oedematous. Not only do the cells become abnormally enlarged, but not infrequently cell divisions also occur. Cell division in the upper epidermis is exemplified in Figure 4, while Figure 5 represents a similar condition in the lower epidermis. A division of the cells in the spongy mesophyll is represented in Figure 3, in which case the elevation extended from the lower surface of the leaf. The cells of the palisade tissue exhibit the same phenomenon (Fig. 2). The thickness of the oedematous portion of the leaf (Fig. 6) may, in extreme cases, be more than double that of the normal tissue. At the time when the tissue begins to brown the plastids in these giant cells are disorganized. Since other cells have ruptured, the entire diseased area becomes shrunken and dried, as shown in Figure 7.

Various explanations have been given from time to time to account for the production of oedema. Giant cells, such as have been described, have been found to occur in connection with insect galls. Woods (1) notes that thin-walled oedematous cells are formed in carnations as a result of the puncture by aphids; and in such event the possible acid conditions are to be considered (11). Both Sorauer (2) and von Schrenk (3) have shown that intumescences may be caused by spraying leaves with copper salts. Various investigators, among whom are Sorauer (4), Atkinson (5), Prillieux (6) and

Noack (7), hold to the general view that the disease is due to impaired transpiration. Sorauer (8) was the first to attribute the cause to abnormal elevation of temperature, together with excessive water supply. He finds that weak light or semi-darkness favors the accumulation of water in the tissues, in that reduced illumination lowers assimilatory activity, and swollen tissue results. On the other hand, Viala and Pacottet (9) believe that brilliant light is a prepotent cause, basing their judgment on the fact that grape leaves directly under the glass in green houses were diseased, while those growing in shade or diffused light were normal. The various workers recognize of course, that these are really the remote causes. For this reason an adequate explanation must describe the chain of events within the tissues themselves.

It is not improbable that the turgor of the giant cells exceeds that of the normal cells. An attempt to discover any such difference in turgor by the plasmolytic method resulted in the detection of no appreciable variation. Both diseased and normal tissues were slightly plasmolyzed by a half-normal solution of potassium nitrate. It may be noted, however, that the leaves examined were somewhat chlorotic and moribund. Again, there appears to be reason for believing that the oedematous condition may be brought about by the imperfect removal of photosynthates. If the carbohydrates were only partially translocated in certain areas the increased concentration of sugars in the cell-sap might tend toward oedema. It is not improbable that a difference in the oxidase content of the healthy and diseased tissues, as has been shown by Bunzel (10) to characterize the curly top disease of beets, is contributory toward inducing this condition. Both of these possibilities are worthy of experimental consideration.

Fisher (11) argues that the development of oedema is due to the increased affinity of the colloids of the tissues for water.[1] This may be brought about by the accumulation of acids, as a result of their abnormal production or inadequate removal. The oedematous tissue of ceará seems to afford some evidence for the truth of this contention. Attempts to determine the acidity of normal and abnormal tissue revealed an easily perceptible difference, the diseased tissue being the more acid. Phenolphthalein was used as an indicator in solutions rendered slightly alkaline by a 1% solution of sodium hydrate. Bits of healthy and abnormal tissue of approximately equal size were macerated in drops of the test solution. Although both kinds of

[1] On this point see critique by Gies (12).

tissue give an acid reaction, yet the oedematous tissue is capable of neutralizing the solution while an equal amount of healthy tissue, macerated in the same quantity of alkaline solution, does not produce this change.

In this preliminary note, it is desired only to call attention to the presence of this interesting disease. We should be glad to receive any information regarding its occurrence from those who have observed ceará in the field. At present we are not aware that the plant is known to suffer from this physiological disorder when growing in its natural habitat. However, the disease well merits further observation and study.

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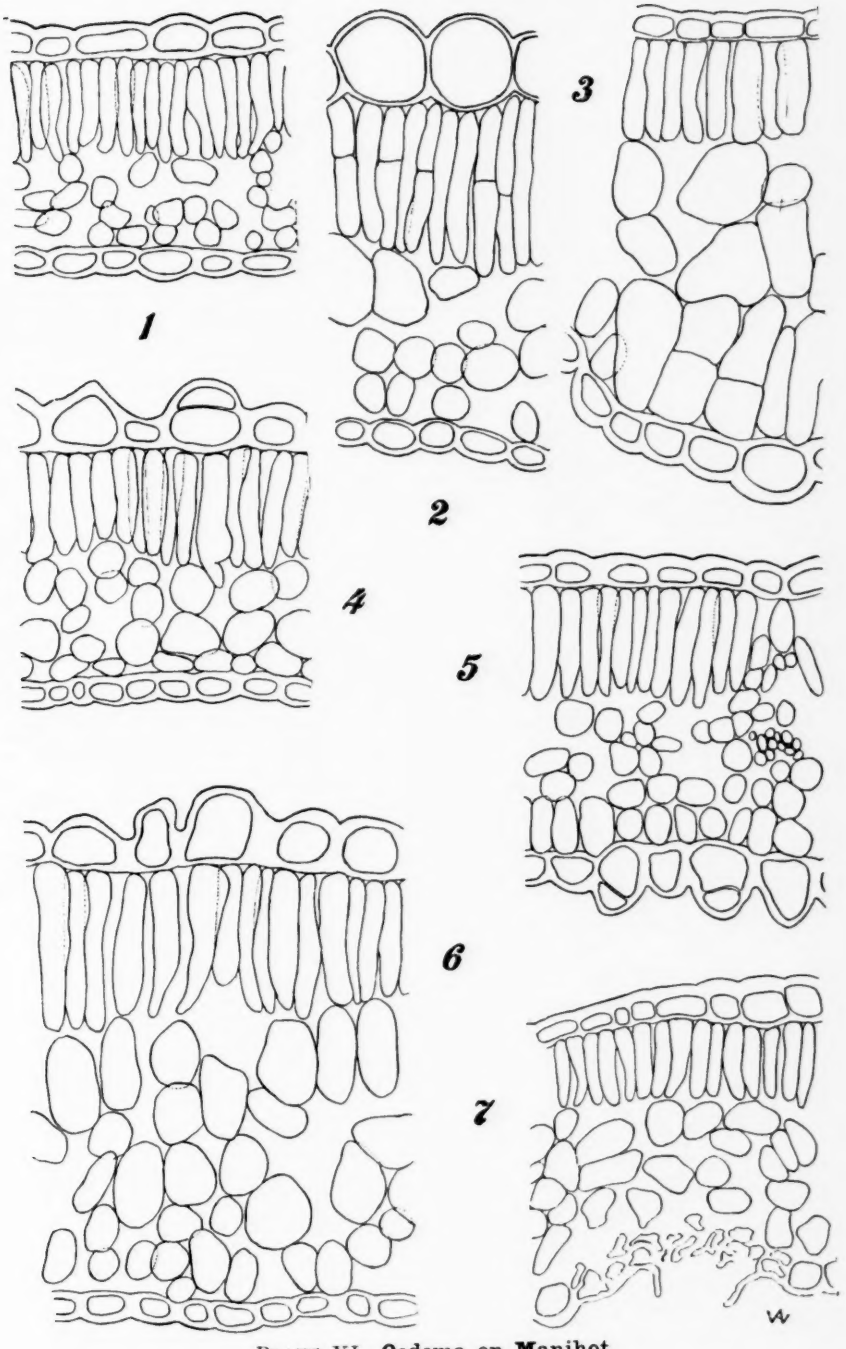

PLATE XI—**Oedema on Manihot**

NOTES ON HAWAIIAN FUNGI. I. GIBELLULA SUFFULTA n. sp.

ALDEN T. SPEARE

(WITH PLATE XII)

Since coming to Hawaii it has been the writers pleasant duty to investigate the fungus flora of the sugar cane insects and being interested in entomogenous fungi in general, it was with great pleasure that he first looked upon the above fungus which is most interesting morphologically and in many ways quite unique.

The specimens were collected by Mr. O. H. Swezey, Assistant Entomologist H. S. P. A. Experiment Station, on fern fronds, in the foothills of the Kohala mountains near Wiamea on the island of Hawaii, but the identity of the host was not revealed until the specimens were brought into the laboratory, and then only when by chance four parallel legs were seen protruding from the mummified, shapeless, fungus-covered body of the host. This immediately indicated that the creature could not be an insect and was some sort of a spider.

When the fruiting stalks (or synnemata) alone are viewed with the naked eye, they have a decided pink tinge, but when examined under a hand lens the stalks themselves appear buff-colored and the spore heads alone have the pink tinge. The mummified body of the spider appears lemon-buff colored.

Several mounts of the fungus in young and old stages were prepared in glycerine-eosin and examination revealed that we had here a fungus quite peculiar to spiders but with unique specific characters.

The synnemata are made up of more or less compact, parallel, septate hyphae which now and then give rise to lateral branches which develop into the sporophores. The origin of these sporophores and the peculiar manner in which they are borne is worthy of comment.

Certain hyphae on the surface of the synnema become bowed out and usually a bud appears on them at a point farthest away from the synnema (Plate XII, Figs. 2a and 16). This bud develops into the sporophore which at maturity, if taken by itself, would have to be placed in the genus Sterigmatocystis. The bud elongates and three cells (sometimes four or five) are cut out by septa (Fig. 19). The ultimate cell gives rise to the spore-bearing part of the sporophore, the middle cell forms the main part of the stalk of the latter and at its base gives rise to a system of anchoring hyphae which will be spoken of later. The lower or proximal cell of the young sporophore does little but lengthen, and grow slightly in diameter altho occasionally it gives rise to anchoring hyphae also. If there are four or five cells instead of three cut out at first we find septa in the main stalk of the mature sporophore but this is not usual (Figs. 5, 6.)

The spore-bearing part arises as follows: The distal portion of the ultimate cell swells gradually and gives rise as it swells, and even before, to buds which grow out to form basidia (Figs. 17, 18, 19) but these buds do not appear simultaneously (Fig. 17) and oftentimes as a result we find spores being formed on one part of the young head while basidia are being formed on other parts. The basidia give rise to a number of sterigmata which also in turn give rise to spores.

Figure 4 shows a partly crushed head of a mature sporophore. First at the lower end of the figure we see part of the main stalk of the sporophore, then next in order the narrow neck of the end cell of the latter with its swelled head, and from this head we see basidia arising and from the ends of the basidia sterigmata and finally, from the tips of the sterigmata, the spores. There are 20–40 basidia on a mature head (Fig. 7) and 10–30 sterigmata on each basidium (Fig. 9). In Fig. 8 we see that the cell walls of the sterigmata are very much thickened where the spores are borne. Just what functions these thickenings may have I have not been able to determine, yet they appear very regularly on all of the sterigmata. Possibly they have something to do with spore abjunction.

The middle cell or cells of the young sporophore grow rapidly in length and diameter and form around themselves remarkably thick walls, but as we have already seen the end cell of the young sporophore grows very little in diameter and but little in length. Consequently where the ultimate cell joins the middle cell there is a noticeable shoulder (Figs. 3, 4) caused in part by the greater diameter, and in part by the thicker wall of the middle cell. Apparently this place of joining is rather weak, for on old sporophores we find that the complete spore head has dropped off at this place. This may be a method of spore dissemination.

The lower cell of the young sporophore does little but increase in length, and slightly in diameter. In Figures 3 and 19 we see a very young and a mature sporophore side by side. The corresponding cells in each case are lettered the same.

Near the septum dividing the middle from the lower cell of the sporophore, and arising chiefly from the upper but sometimes from the lower cell, are two or more branches. In Figure 18 we see one branch well developed and, exactly opposite, the beginning of another. These branches grow toward the synnema, and in turn become septate and branched. Their ultimate branches enter the mass of hyphae of the synnema so that a mature sporophore has a branched system of hyphae near its base which cause it to stand out conspicuously from the synnema (Figs. 2, 12).

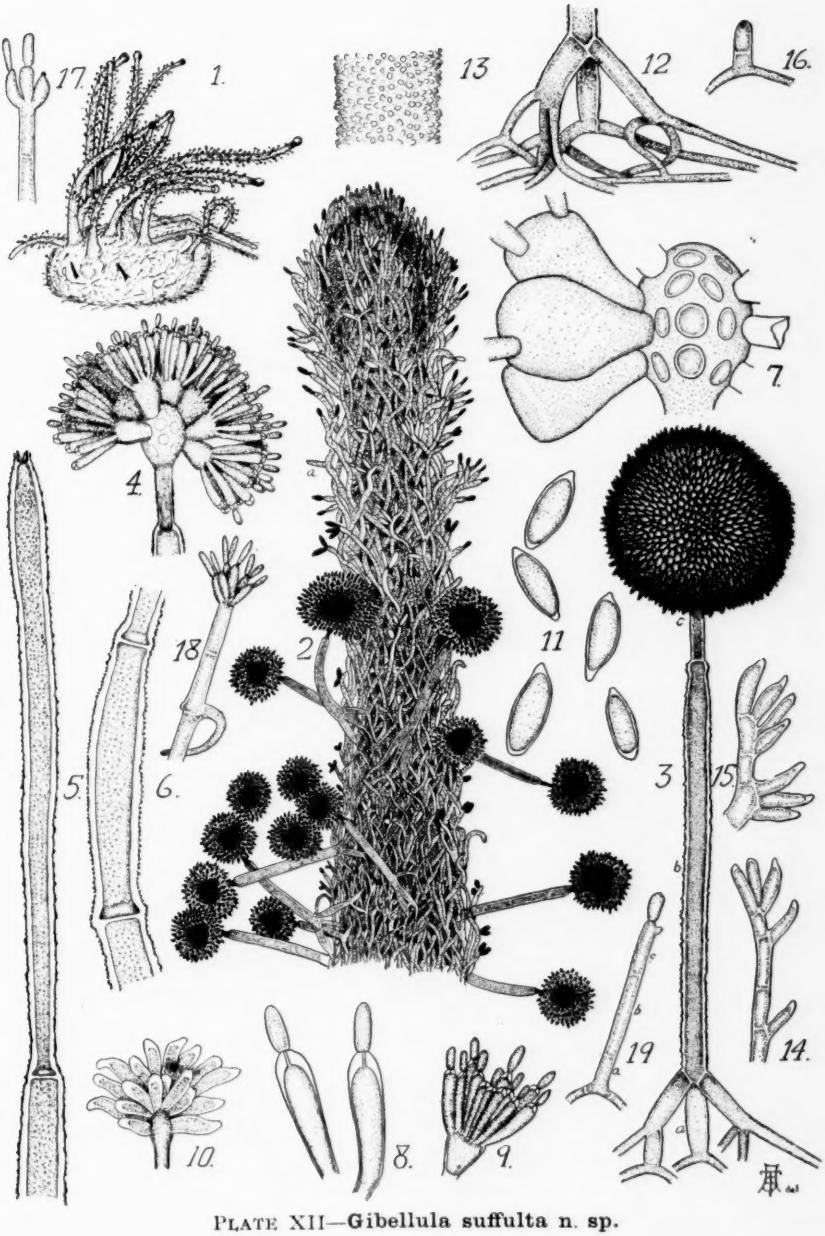

PLATE XII—**Gibellula suffulta n. sp.**

This system of branched hyphae and the thick walls of the stalk cells are doubtless of value in extending the sporophores out as far as possible from the synnema and supporting them at right angles to the latter. It is quite obvious that if neither of these conditions were present the sporophores would bend down under the heavy weight of the spore heads.

The spore-bearing area is not necessarily restricted to the sporophores which arise from the synnemata. Oftentimes there are sporophores scattered over the mummified body of the spider and spores are also borne on irregular shaped basidia arising directly from the surface of the synnema (Figs. 2, 14,15).

This fungus readily falls into the genus Gibellula but it seems to have characters that give it specific rank. Because of the peculiar system of hyphae at the base of the sporophores I have chosen to call it *suffulta*.

Gibellula suffulta, n. sp.

Entirely buff colored, spore heads pinkish, synnemata rising up 4-7 mm. high, 80-100 mm. in diameter, made up of compact septate hyphae; sporophores 150–180μ long, 6-8μ in diameter, bearing at the tip a globose head carrying basidia, sterigmata, and spores. Total diameter of spore head 54-68μ. Diameter of globose head bearing basidia etc. 8.5-11μ. Basidia 12μ long, 7.5μ wide. Sterigmata 9-12μ long, 3μ wide at base, 5μ wide at tip. Wall thickened 4-5 times at tip.

Spores sub-hyaline to hyaline, ovoid, 6–7.5μ long, 2-3μ wide; wall thickened at either end. On an unknown species of spider. Wiamea, Hawaii, Dec. 1911.

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DESCRIPTION OF PLATE XII

FIG. 1. Mummified body of spider with fruiting stalk, x 3.5.
FIG. 2. Tip of a fruiting stalk, x 125.
FIG. 3. Mature sporophore, x 425.
FIG. 4. Partly crushed head of mature sporophore, x 575.
FIGS. 5-6. Portion of stalk of sporophore, Fig. 5, x 575; Fig. 6, x 850.
FIG. 7. Crushed head of sporophore showing method of insertion of basidia, x 1700.
FIG. 8. Sterigmata, x 1700.
FIG. 9. Basidia with attached sterigmata, x 850.
FIG. 10. Irregular spore head, x 850.
FIG. 11. Spores, x 1700.
FIG. 12. Base of sporophore with its branched anchoring hyphae, x 600.
FIG. 13. Surface of stalk of sporophore, x 1700.
FIGS. 14-15. Irregular sporogenous hyphae, x 850.
FIG. 16. First stage of young sporophore, x 850.
FIGS. 17, 18, 19. Young sporophores, x 850.

THE COMPARATIVE SUSCEPTIBILITY OF CRUCIFEROUS PLANTS TO PLASMODIOPHORA BRASSICAE

G. C. CUNNINGHAM

Since the publication of Woronin showing that *Plasmodiophora brassicae* is a parasite of cabbages and some other crucifers, the statement that most crucifers are susceptible to its attack has gradually found place in literature. Some writers have gone so far as to claim that families other than the crucifers are susceptible. Marchand[1] describes a disease of certain varieties of the *Cucurbitaceae*, *Umbelliferæ* and *Polygonaceae* which he claimed to be caused by *Plasmodiophora brassicae*. Grignan[2] states, however, that a subsequent examination of these plants showed that the malformations on the roots were caused by nematode worms, *Heterodera radicicola*. Similar galls on other plants are caused by nematode worms and it is possible that such injuries are at times mistaken for club root. A review of the literature at hand shows that there are seven native genera which are susceptible to the attack of *Plasmodiophora brassicae*.

Among those who have added to our knowledge on this phase of the question, mention should be made of the work of Woronin[3], Saccardo[4], Zopf[5], Sorauer[6], Frank[7], Halsted[8], Ravn[9], and Appel[10].

According to Halsted, who has shown the wide range of susceptibility among the *Cruciferae*, the following seven species are affected by this disease, but certain of them only rarely. *Brassica oleracea* (cabbage), *Brassica rapa* (turnips), *Raphanus sativus* (radish), *Sisymbrium officinale*, *Iberus umbellata*, *Matthiola incana* (stock), *Capsella bursa-pastoris* (Shepherd's purse). The last three are rarely affected.

Appel and Werth ([10]) examined a number of the crucifers growing on soil which was known to be infected with the club root organism

[1] Marchand, E. F. L. Le Plasmodiophora Brassicae Woronin, parasite du melon, du céleri et de l'oseille, épinard. Compt. Rend. Acad. Sci. (Paris) **150**: 1348–1350. 1910.

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and have extended the above list by adding : *Erysimum strictum*, *Sisymbrium austriacum*, *Sisymbrium strictissimum*. Ravn (l.c.) has compiled a list including nineteen genera, eight of which are known to be susceptible to the attack of *Plasmodiophora brassicae* in Denmark. He added *Thlaspi arvensis* and *Cheiranthus cheiri* to the list of known hosts.

There is however very little literature bearing upon the range and comparative amount of susceptibility among the *Cruciferae*. This is partially due to the fact that much information is covered up by the statement that most crucifers are susceptible to the disease. This statement, while probably true, has little recorded information to support it, as shown by the comparatively limited lists just given.

A piece of land near Burlington, Vt., belonging to Mr. H. S. Merrihew, a market gardener, upon which varieties of *Brassica oleracea* have been grown commercially for a number of years, has become so badly infected with club root that such plants can not be profitably raised on it. In the spring and summer of 1911 use was made of this field to study, among other problems, the question of comparative susceptibility of cabbages and other crucifers to the attack of *Plasmodiophora brassicae*. For this purpose as many varieties of crucifers as possible were obtained from various sources and sown upon the infected soil. Each variety of seed was placed in several different parts of the field so that by averaging the results the factor of variations in soil conditions might be eliminated as far as possible. The cabbage (*Brassica oleracea* var. *capitata*) and cauliflower (*Brassica oleracea* var. *botrytis*) were started in cold frames and after three or four weeks were transplanted to the field ; in case of other varieties, the seeds were sown directly in the field.

At irregular intervals throughout the season the plants were examined for the appearance, nature and extent of the disease. The plants were carefully dug up with a fork rather than pulled up, as it was found that many clubs on lateral roots were broken off by the latter method, this being particularly true of the radishes and turnips. Notes were made under the following heads :

1. General appearance of plants ; 2. number examined ; 3. number badly clubbed ; 4. number slightly clubbed ; 5. number showing free or adventitious rootlets ; 6. in the case of cabbages the number and weight of solid saleable heads was determined at the end of the season.

The results are summarized in two tables. Table I shows the comparative susceptibility of the thirteen varieties of cabbages tested. The arrangement is in the order of their susceptibility, beginning with the badly clubbed varieties. Table II shows the comparative suscepti-

bility of the different crucifers which were included in this experiment. These plants are also arranged in the order of their susceptibility, beginning with the badly clubbed varieties. It will be seen that this year's test indicates that some varieties are considerably more resistant than others.

TABLE I. RESISTANCE OF DIFFERENT VARIETIES OF CABBAGES TO PLASMODIOPHORA BRASSICAE

Variety of cabbage	Total number examined	Percentage not clubbed	Percentage clubbed	Percentage badly clubbed	Percentage slightly clubbed
Mammoth Rock Red	275	.0	100.0	97.1	2.9
Perfection Savoy	279	.0	100.0	92.5	7.5
American Savoy	249	.8	99 2	80.0	19.2
Dark Red Erfurt	238	1.2	98.8	86.6	12.2
All Seasons	654	2.7	97.3	88.8	8.5
Henderson's Early Summer	356	5.6	94.4	72.0	22.5
Early Winningstadt	339	5.6	94.4	63.7	30.7
Charleston	304	5.9	94.1	74.6	19 5
Volga	546	8.8	91.2	82.0	9.2
Early Jersey Wakefield	274	9.4	90.6	47.5	43.1
Large Late Flat Dutch	486	9.9	90.1	62.6	27.6
Stone Mason	180	14.4	85.6	65.6	20.0
Hollander	541	26.5	73.5	52.6	20.9

TABLE II. SUSCEPTIBILITY OF VARIOUS CRUCIFERS TO PLASMODIOPHORA BRASSICAE

Name of plant examined	Number examined	Percentage not clubbed	Percentage clubbed	Percentage badly clubbed	Percentage slightly clubbed
Brassica oleracea var. botrytis (cauliflower)*	934	0	100	100	0
Brassica alba (white mustard)*	503	0	100	100	0
Brassica arvensis (charlock)	214	0	100	100	0
Brassica rapa (southern curled turnips)*	584	0	100	100	0
Brassica pe-tsai 17593**	18	0	100	100	0
Camelina dentata	60	0	100	100	0
Neslia paniculata	41	0	100	100	0
Brassica campestris (rutabaga from India) 26926**	274	4.3	95.7	95.7	0
Brassica oleracea var. caulorapa (kohl rabi)*	1406	4.7	95.3	79.7	15.6
Thlaspi arvense (field penny cress	36	5.5	94.5	86.1	8.4
Brassica oleracea (cabbage) 26564**	207	5.8	94.2	94.2	0
Brassica oleracea var, capitata (cabbage)*	4721	6.9	93.1	74.4	18.7
Brassica oleracea var. acephala (collards)*	3129	7.2	92.8	66.1	26.7

* Cultivated plants which are ordinarily grown in fields and gardens.

** Seeds which have been lately introduced into the United States by the Bureau of Plant Industry; figures indicate B. P. I. accession number.

Name of plant examined	Number examined	Percentage not clubbed	Percentage clubbed	Percentage badly clubbed	Percentage slightly clubbed
Erysimum cheranthoides (worm-seed mustard)	124	8.1	91.9	13.6	78.3
Brassica sp. (cabbage from Abyssinia) 25682**	706	9.3	90.7	90.7	0
Camelina microcarpa	19	10.5	89.5	0	89.5
Brassica oleracea var. botrytis*	3137	11.5	88.5	63.2	25.3
Brassica oleracea var. gemmifera (brussels sprouts)*	376	13.6	86.4	86.4	0
Erysimum parviflorum	14	14.3	85.7	85.7	0
Brassica napus (rape)*	2288	16.3	83.7	32.5	51.2
Brassica oleracea 21033**	76	18.4	81.6	81.6	0
Sisymbrium altissimum (tumbling mustard)	48	20.8	79.2	52 1	27.1
Brassica (large flat green turnips) 25592**	89	22.5	77.5	72	5.5
Hesperis matronalis (Dame's violet)	58	31.1	68.9	13.8	55.1
Raphanus sativus (round black Spanish radish)*	1020	34.1	65.9	56.1	9.8
Radish (from west China) 30194**	320	34.4	65.6	59.4	6.2
Brassica oleracea var gemmifera (brussels sprouts)	195	36.4	63.6	28.2	35.4
Raphanus sativus (several varieties)*	437	41.4	58.6	38.7	19.9
Capsella bursa-pastoris (Shepherd's purse)	14	42.7	57.3	57.3	0
Lepidium apetalum	21	42.7	57.3	14.6	42.7
Brassica oleracea var. caulorapa (kohl rabi)	189	46	54	29.1	24.9
Raphanus sativus (everlasting radish) 10399*	483	46.2	53.8	0	53.8
Brassica oleracea var. acephala (kale)	186	46.3	53.7	14.4	39.3
Raphanus sativus (radish) 30193**	580	46.6	53.4	53.4	0
Raphanus sativus (radish) 25593**	176	46.6	53.4	43.7	9.7
Arabis alpina	21	47.6	52.4	9.6	42.8
Erysimum aspernum	16	50	50	31.3	18.7
Brassica napus	264	50.8	49.2	10.2	39.0
Raphanus sativus (White-tip radish)*	471	54.0	46	35.4	10.6
Lobularia maritima (sweet alyssum)	18	55.6	44.4	16.6	27.8
Conringia orientalis (hare's ear mustard)	191	59.2	40.8	13.6	27.2
Brassica nigra (Black mustard)	31	61.3	38.7	25.8	12.9
Lepidium campestre	57	61.4	38.6	21.1	17.5
Raphanus sativus (Giant radish)*	672	83.1	16.9	11.3	5.6
Brassica campestris (cultivated turnips)	484	88	12	5.2	6.8
Brassica rapa (Petrowski turnips) 22755**	172	90.7	9.3	6.4	2.9

* Cultivated plants which are ordinarily grown in fields and gardens.

** Seeds which have been lately introduced into the United States by the Bureau of Plant Industry. ; figures indicate B. P. I. accession number.

Name of plant examined	Number examined	Percentage not clubbed	Percentage clubbed	Percentage badly clubbed	Percentage slightly clubbed
Matthiola bicornia	88	92.1	7.9	0	7.9
Barbarea stricta	13	93	7	0	7
Brassica campestris (uncultivated turnips)	217	94.5	5.5	0	5.5
Brassica rapa	237	98.7	1.3	0	1.3
Brassica rapa (Early White Mullen turnips)*	608	98.9	1.1	.8	.3
Lepidium sativum	107	100	0	0	0
Draba androsacea (Whitlow grass)***		100	0	0	0

* Cultivated plants which are ordinarily grown in fields and gardens.
*** These plants form masses of tufted moss-like vegetation lying close to the surface of the ground. Two boxes have been growing upon infected soil for one year and have never shown clubbing during that time.

From the comparison made in the two tables it is evident that there is a wide range of susceptibility among the *Cruciferae* not only among the different genera but among the species within the genera and to an equal extent among the different varieties of the same species. This is brought out clearly by the fact that the different species of the same genera are widely scattered throughout the table. One variety of *Brassica oleracea* shows 100 per cent badly clubbed while *Brassica rapa* shows 1.1 per cent clubbed. A similar condition exists among the Lepidiums where *Lepidium campestre* shows 38.7 per cent clubbed as compared with *Lepidium sativum* which was entirely free from clubs.

A similar variation of susceptibility exists among the different varieties or strains of the same species as indicated by the scattering of the varieties of *Brassica oleracea* and *Raphanus sativus*. This is indicated in Table I showing the wide range of susceptibility among the different varieties of cultivated cabbage, where the Mammoth Rock Red variety shows a susceptibility of 100 per cent as compared with the Hollander with a susceptibility of 73.5 per cent. The same conditions exist among the varieties of *Raphanus sativus* (radish) as seen in Table II, where Round Black Spanish radish shows a susceptibility of 65.6 per cent as compared with Giant radish which shows 16.9% susceptibility. Similar variations in susceptibility exist among the different varieties of other species.

The wide range of susceptibility among the species and varieties offers a hope that among the cabbages, turnips, and radishes, the most generally cultivated crucifers, there may be some varieties more resistant to this parasite than are the ones commonly cultivated.

AGRICULTURAL EXPERIMENT STATION,
UNIVERSITY OF VERMONT,
BURLINGTON

A NEW LEAF RUST OF PEACH

S. HORI

(WITH PLATES XIII AND XIV)

When I visited the horticultural experimental field of our Station at Okitsu in the Province of Suruga, November 6, 1910, my attention was attracted by the fact that the leaves of the peach trees, both in the nursery and in the field, were suffering severely from rust and bore snowy white sori on their under surface. On microscopical examination it was found that the fungus was a species of Puccinia entirely different from either *P. pruni-spinosae* Pers. (*P. pruni* Pers.) or *P. cerasi* (Bereng.) Cast., the only species of the genus hitherto known as parasitic on the peach and its allies.

According to Sydow[1], *P. cerasi* is found only in Europe and *P. pruni-spinosae* is widely distributed throughout the world. The latter has been found in Japan on *Prunus buergeriana* Miq., *P. mume* S. and Z., *P. americana* var. *ansu* Maxim., *P. grayana* Maxim., but not yet on the peach. Since the rust in question is due to a new species of Puccinia and is characterized by its white teleutosori, I have given it the pathological name, white leaf rust of peach, to distinguish it from the brown leaf rust (*P. pruni-spinosae*) and yellow leaf rust (*P. cerasi*).

Symptoms of the disease. The disease seems to make its first appearance about the end of July or the beginning of August. When the conditions are favorable, it continues to spread until the fall of the leaves in late autumn. The disease is usually confined to the leaves. At first small rounded purplish brown spots with indistinct margins appear, scattered in great numbers over the upper surface of the affected leaf. Later the center of each spot changes to a light yellowish brown or ocher color and the spot itself becomes polygonal in shape and 1-2 mm. in diameter, with a distinct margin surrounded by the purplish brown colored part. In some varieties of peach, the spots assume from the very beginning a light yellowish brown or ocher color with distinct margin. With the lapse of time the spots tend to coalesce into large irregular areas. On the under surface of the leaf spots the epidermis swells up hemispherically and subsequently bursts and a light brown or cinnamon colored powder makes its appearance. From about the end of October numerous small snowy white pustules of round form and somewhat sticky consistency appear between the cinnamon colored powdery pustules, the uredo-

[1]Sydow, P. and H. Monographie Uredinearum, I : 484-487. 1904.

sori. In December only these white pustules are apparent on the affected leaves.

The diseased leaves soon become yellowish brown and fall off at the slightest touch. Since the disease appears at first on the old leaves and gradually spreads to the upper and younger ones, the severely affected peach trees are defoliated very early and only a few leaves persist near the top of the branches. Hence the fruit is poor in quality and the wood is not properly matured especially with seedlings.

In the summer and autumn of 1910, there was in Japan an unusually long continued rainy period. This condition must undoubtedly have favored the severe outbreak of the disease that year and the specimens received from several places through the courtesy of friends, prove that the disease occurred not only in Okitsu but also in several other places in Japan. The peach trees in the nursery where nitrogenous manure had been applied in comparatively large quantities to advance growth, were more severely subject to the rust than those in the field.

Morphology of the fungus. The white leaf-rust of peach trees is caused by the attack of a species of Puccinia hitherto unknown. Since the fungus is pathogenic to peach trees I have given it the scientific name *Puccinia pruni-persicae*. The description is as follows :

Puccinia pruni-persicae n. sp.

Uredosori hypophyllous, cinnamon colored, rounded, surrounded by the ruptured epidermis, small, scattered or crowded, pulverulent ; spores elliptical, obovoid, oblong, 30-42x14-20μ ; apex thickened, bluntly conical, upper part smooth and brown colored, lower part coarsely echinulate and almost colorless, darker at the apex, contents coarsely granular, colorless ; pedicels short, colorless, deciduous ; paraphyses numerous, between the spores, spherical at top, 12-15μ broad by 45-70μ long, smooth, with very little granular contents ; pedicels irregularly curved.

Teleuto-sori hypophyllous, snowy white, rounded, small, scattered or crowded, often confluent, soon ruptured, compact, somewhat sticky ; spores long fusiform, oblong, often clavate, constricted at the septum, 32-44 x 12-14μ, apex conically attenuated, base truncated, epispore uniformly thin, smooth, colorless, contents finely granular ; pedicels persistent, long, colorless, 30-60 x 6-8μ. Spores soon germinate, producing from each cell a long 2-3 septate promycelium, 50-55 x 6μ ; sporidia obovoid 7-9 x 6-7μ.

HAB.—On living leaves of *Prunus persica* S. and Z. var. *vulgaris*

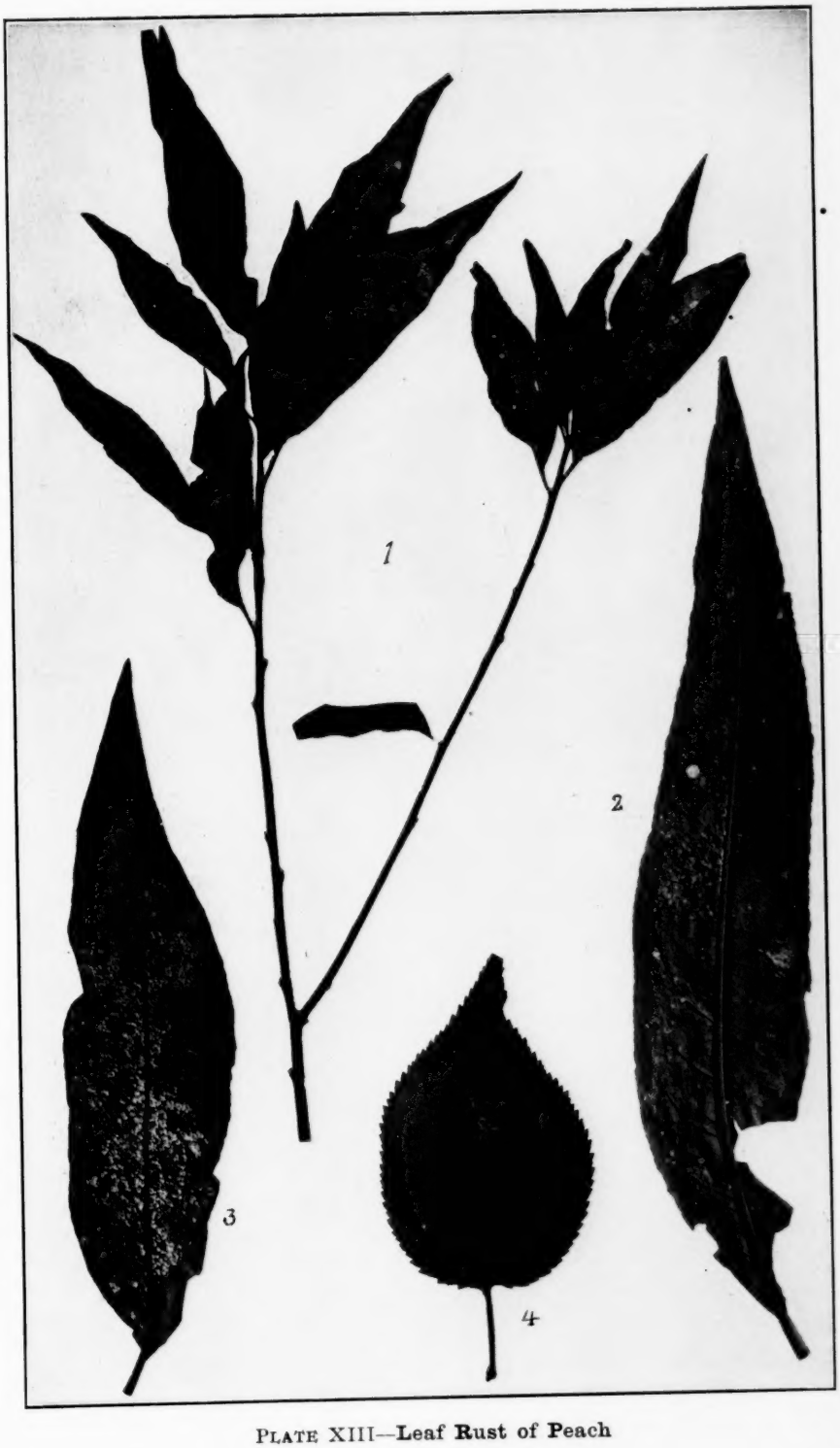

PLATE XIII—**Leaf Rust of Peach**

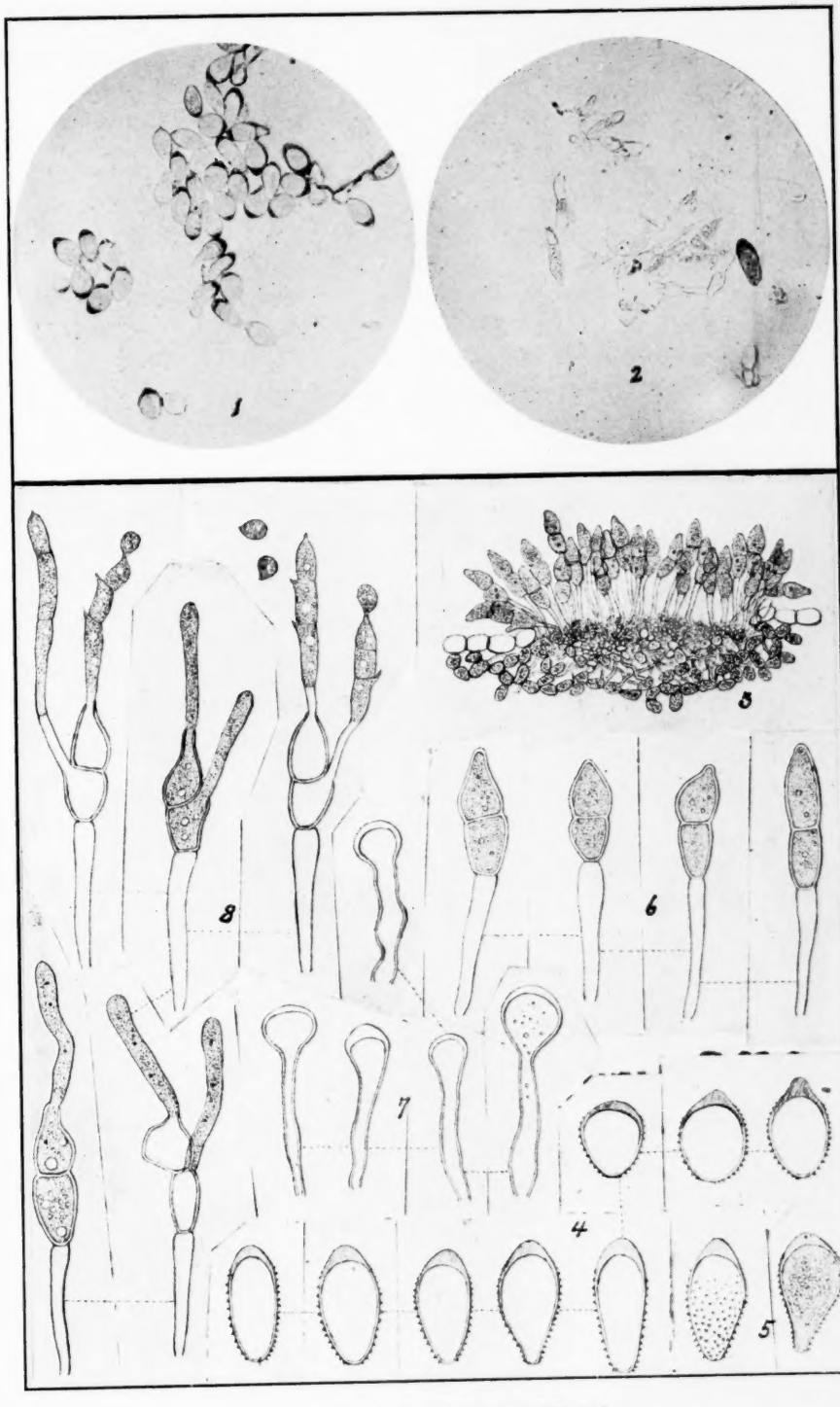

PLATE XIV—**Leaf Rust of Peach**

Maxim. Okitsu, Prov. Suruga (II,III. Nov. 8, 1910. S. Hori ; Nov. 30, 1910. K. Kawai) ; Dogo, Prov. Iyo. (II. Oct. 10, 1906. T. Nishida) ; Wakayama, Prov. Kii (II. Nov. 24, 1910. U. Bokura) ; Kochi, Prov. Tosa (II,III. Dec. 27, 1910. T. Yoshinaga) ; Maebashi, Prov. Kozuke (II. Sept. 30, 1910. K. Yoshino) ; Hojomura, Prov. Bijen (II. Aug. 15, 1909. M. Kasai) ; Fujimimura, Prov. Suruga (II. Aug. 18, 1910. S. Tsurta) ; Akashi, Prov. Harima (II. Oct. 1903. T. Yamaguchi).

It is easily distinguished from *P. pruni-spinosae* by the absence of mesospores, white teleutosori, smooth and colorless teleutospores ; from *P. cerasi* by the white teleutosori, smooth and colorless teleutospores and presence of paraphyses. The uredo stage of the present fungus until the discovery of its teleutostage, was erroneously known as the uredostage of *P. pruni-spinosae*, but close microscopical observations revealed the fact that the uredo-spores of *P. pruni-spinosae* are all thickened at the apex and so-called mesospores are absent.

Teleutospores soon germinate at maturity and produce sporidia. Whether it produces aecidia on some species of Anemone, as in *P. pruni-spinosae*, or on entirely different plants, requires further investigation.

Prevention. Last year the season was too late to carry out experiments for prevention, but spraying two or three times with a dilute solution (10-20 times) of lime sulphur mixture will be sufficient to prevent the disease.

EXPLANATION OF PLATES

PLATE XIII. Fig. 1. Upper part of the severely rusted branch of seedling peach (much reduced). Fig 2. Uredo-sori with a few teleutosori on the under surface of the affected leaf (slightly reduced). Fig. 3. Teleuto-sori on the under surface of the affected leaf (slightly reduced). Fig. 4. *Puccinia pruni-spinosae* on the leaf of *Prunus mume* S. and Z.

PLATE XIV. Fig. 1. Uredospores (Zeiss 2 x DD); Fig. 2. Teleutospores (Zeiss 2 x DD). Fig. 3. Section of teleuto-sori (Zeiss 2 x DD). Fig. 4. Uredospores (Zeiss 2 x F). Fig. 5. Uredospores showing the contents and surface (Zeiss 2 x F). Fig. 6. Teleutospores (Zeiss 2 x F). Fig. 7. Paraphyses (Zeiss 2 x F). Fig. 8. Germinated teleutospores (Zeiss 2 x F).

IMPERIAL CENTRAL AGRICULTURAL EXPERIMENT STATION,
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DOES THE POTATO SCAB ORGANISM[1] SURVIVE PASSAGE THROUGH THE DIGESTIVE TRACT OF DOMESTIC ANIMALS?

W. J. MORSE

(WITH PLATE XV)

It is a well known fact that potato scab will persist in infected soils for an undetermined period, at least 25 years or more.[2] It has also been demonstrated repeatedly that application of fresh stable manure to the soil immediately before planting tends to increase the amount of scab on the resulting potato crop. This is generally explained as being due to chemical changes in the soil, as a result of the application of the manure, which favor the development of the causal organism. The evidence appears to be quite conclusive on this point. It is also generally conceded that the principal source of infection of previously uncontaminated soils is the use of scabby seed tubers.

So far as the writer has been able to discover there is no published data regarding the manure itself as a possible or probable source of infection. It is quite conceivable, where uncooked potato peelings are allowed to become mixed with the manure, or scabby potatoes are thrown into pig pens or barn lots to be eaten there by the animals, that the litter and manure in such cases will become an active agent in spreading the disease. The question arises, however, whether or not it is safe to feed uncooked potatoes or potato refuse to animals in stalls provided due precautions are taken against allowing any of the uneaten portions to become mixed with the litter and manure. In other words, are the germs of potato scab able to pass through the digestive tract of herbivorous animals and still be viable? It was to obtain data upon this point that the following experiments were made. In certain northern potato growing states, notably Maine, Michigan, Wisconsin, Minnesota, etc., this is an important question. In these states a varying amount of uncontaminated virgin soil is yearly being cleared and planted to potatoes. The value of this soil for potato

[1] In a paper read at the last meeting of the American Phytopathological Society Cunningham has suggested that the potato scab organism, *Oospora scabies*, should be more properly referred to the genus Streptothrix of the Mycobacteriaceae (see p. 97 of this volume.) The writer from his studies of this organism, extending over several years, is convinced that this is the correct position to take.

[2] Jones, L. R., and Edson, A. W. Vt. Exp. Sta. Rep. 14 : 232. 1901.

growing in years to come will depend largely upon how fully the germs of scab and other soil-inhabiting potato pathogens are excluded.

Two different experiments were conducted, one in 1909–10 and the other in 1911. The horse and the cow were the animals used. Of necessity the experiments were conducted on a small scale, only one animal of each kind being used in each experiment.

Experiment I. The potatoes used for feeding were exceedingly scabby—the majority being one solid mass of scab spots. Both animals were of mature age and of medium size. The feeding began on November 25, 1909. The horse received 30 pounds of potatoes per day, a part at each feed, while in the same way the cow was fed 40 pounds per day. On the fourth day all of the solid excrement voided by the horse was collected in sterilized tin pails without allowing it to touch anything else after leaving the animal's body. The cow's manure was collected in the same way on the sixth day after the feeding was begun. The pails were provided with tightly fitting covers and after being filled were stored in a cool cellar, properly protected from outside contamination and from drying out.

The ability of the manure to produce scab was tested in the following manner. Ten-inch flower pots were thoroughly scrubbed to remove all traces of dirt. Next they were immersed in a disinfecting solution of formaldehyde for 24 hours, followed by soaking in boiled water to remove all traces of the formaldehyde and then allowed to dry out for several days. Next the pots were partly filled with soil which had been sterilized by two successive steamings of two hour's duration, in small lots, under a pressure of 15 pounds. The two lots of manure were then each divided up among 7 pots and thoroughly mixed with the upper layers of soil with sterile paddles. Two check pots were filled with sterilized soil alone, making 16 pots in all. Next absolutely smooth, medium sized potato tubers were selected and disinfected in the usual way with dilute formaldehyde solution. After drying in the open air for 24 hours, but protected from any possible contamination by scab germs, one of these tubers was planted in each pot, without cutting. The pots were then placed in the greenhouse on a raised platform made of fresh, clean boards which had first been thoroughly treated with formaldehyde solution some days before.

Precautions were taken to avoid any outside contamination of the experimental pots by means of infected soil and they were watered with boiled water till the plants were mature. The resulting tubers were then examined for scab spots with the following results:

	Total number of tubers.	Number smooth.	Number scabby.
Horse manure	61	45	16
Cow manure	91	91	0
Check	16	16	0

Nearly all of the scab spots on the tubers from the horse manure were very minute. Only 4 showed more than 3 small spots. One, however, had 14 spots scattered over the surface.

These results were suggestive but not conclusive, for two serious objections might be raised as to factors influencing them. First the manure was collected the last of November but conditions prevented its use until April 21, following. Second the metal pails used for storage might exert some germicidal effect on the organisms.

Experiment II. In this case a mature horse and a two-year-old heifer were the experimental animals. The horse was fed 15 pounds of potatoes the first day, May 17, 1911, and then 25 pounds per day thereafter. The manure was collected on the fifth day. The heifer was fed 15 pounds the first two days, 23 the next two and 30 pounds per day thereafter. The manure was collected on the ninth and tenth days. This time the manure was stored in sterilized wooden pails and was placed in the pots with the sterilized soil and seed tubers with a delay of only a few days, thus meeting the objections raised regarding the first experiment. Otherwise the conditions of the two experiments were as near alike as they could be made. The tubers used for feeding in the second case were fully as scabby as the first. The pots were emptied and the new tubers examined on September 8.

	Total number of tubers.	Number smooth.	Number scabby.
Horse manure	50	12	38
Cow manure	65	54	11
Check	17	17	0

The accompanying figure shows three tubers from each lot which were selected with a view of representing as nearly as possible the average condition of each. The tubers from the check pots were absolutely smooth and free from scab. Some of the tubers from the cow manure showed several small scab spots each and over 80% of the whole were without spot or blemish. On the other hand over 70% of the tubers produced with the horse manure were scabby, the majority of them being badly infected. Taking into consideration the anatomical differences in the digestive tracts of the horse and cow the relative amounts of scab obtained from the manure of each are about as might be expected.

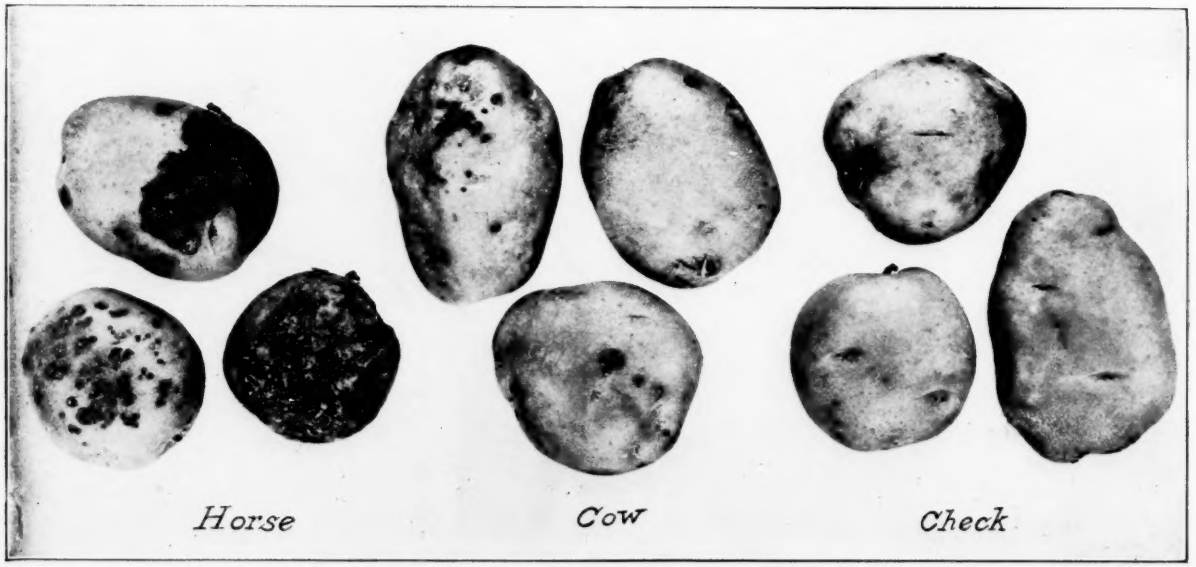

PLATE XV—**Potato Scab Experiment**

Three tubers at left grown in pots where manure used was from horse fed on scabby potatoes; those in middle from cow similarly fed; control at right, no manure. These tubers were fairly representative of the average conditions in each lot.

CONCLUSIONS

The results obtained indicate that the germs of potato scab are able to pass the digestive tract of both the horse and the cow and go into the manure pile without being destroyed, but much more readily in the case of the former than with the latter animal. The manure of horses fed on raw potatoes is very likely to carry the germs of the disease. On the other hand manure from cows fed a moderate quantity of potatoes, which are not excessively scabby, is probably not a serious source of contamination.

MAINE EXPERIMENT STATION,
ORONO, MAINE

A COLLEGE COURSE IN PLANT PATHOLOGY

BRUCE FINK

In statements regarding the best college preparation for phytopathology one rarely finds mention of anything concerning direct study of the subject itself. Usually the student is advised to study bacteriology instead. This is, of course, due to several causes. The technique of elementary laboratory work in bacteriology is well developed and the routine of operation is somewhat standardized. On the other hand, one may search in vain for any general plan for laboratory work in plant pathology. The college teacher of botany seldom knows much about the latter subject and rarely does he or his institution care to introduce such technical work into laboratory and class room. The laboratory work involves a good deal of expense, which should not be incurred unless there is a real demand for the course. For these reasons, and others, it usually seems best that the student in a college of liberal arts should, even if he wishes to prepare for plant pathology, postpone work in this field until he has finished his college course.

But, given a teacher interested in the problems, of horticulture, forestry and agriculture, who is sufficiently acquainted with mycology, plant physiology and ecology, familiar with general laboratory methods in plant pathology and able to recognize the symptoms and effects of plant diseases in the field and to apply remedies, then the only obstacles to giving such a course lie in whatever prejudice plant pathologists may have against such a procedure and in the doubt whether such work should form part of a course in liberal arts, even for students who wish to enter horticulture, agriculture or plant pathology. When we consider the difficulty of finding college teachers who can give the course and the valid argument against offering such technical work in most colleges, it seems probable that college courses in plant pathology will never become common.

The conditions at Miami University which have led to giving a course in plant pathology are peculiar in several ways. The professor of botany must be able to give general and premedical bacteriology. The institution is located in a large village in the midst of an agricultural community, from which a considerable number of farmers' sons enter for preliminary training before going to agricultural schools. Others come to prepare for the study of forestry. A four-year course in elementary agriculture is offered in our normal school, and the young men take botany in the college of liberal arts, some completing majors in the subject.

Like some other colleges, we have had calls from time to time for

young men desirous of taking up work in plant pathology, and it appeared that our conditions were such that we were doing students who were interested in such work positive injustice by not introducing an elementary course in the subject. Consequently, plans were begun two years ago, looking toward offering a four-hour semester course. With the aid of a careful and persevering student, the laboratory and field experiments were tested, a manual was prepared for our own use and more than twenty diseases were selected to form the basis of lectures, reading, and laboratory and field work. The literature of these diseases was gone over carefully in order that we might know the various views regarding their nature and treatment. The course was introduced after the details had been worked out.

Preliminary to the consideration of these diseases, the history of plant pathology is gone over, together with the relations of this branch to other sciences, the causes and the physiology of plant diseases, their symptoms and effects, prevention and cure, methods of infection and resistance and susceptibility. Then each disease is considered in about the following order,—the hosts, symptoms, methods of attack of the causal organism, time and conditions of attack, effects of disease, preventive measures, the history and the distribution of the disease, culture methods, infection methods, general notes and bibliography. The morphology and the physiology of the causal organism are included in the lecture notes, but only such portions as are of special interest to the pathologist are touched in the course, the student being required to read on these unless he has the knowledge required from the year's course in mycology. The diseases considered are wheat rust (two forms), apple rust and cedar rust, potato mildew, apple scab, leaf-curl of peach, chestnut bark disease, pear blight, black rot of cabbage, black rot of apple, downy mildew of grape, black knot of plum, plum pocket, red heart rot of trees, root rot of trees, bean anthracnose, leaf spot of beet, and ring scale of pine. The selection was made on account of importance and local interest of the diseases. Probably no two teachers would select exactly the same diseases, but these include the most important infectious diseases and serve to make the student familiar with the general subject of plant diseases. The selected bibliography averages about twenty five titles for each disease considered, and readings are assigned to the students. The course deals exclusively with phytopathology, mycology being offered separately for as many years as the student desires to give to the subject. The absence of any consideration of physiological diseases will be remedied when further study gives something tangible to present.

The laboratory is thoroughly equipped for work on infectious animal

and plant pathology, and the course alternates with bacteriology, each being given once in two years. The subjects considered in the laboratory work and the field studies are cleaning glassware ; the preparation of about 15 kinds of media ; transferring, isolating, eliminating, and staining bacteria ; chart study of fungi and bacteria : obtaining and germinating fungus spores ; making pure cultures of fungi and bacteria ; inoculations by spores and by dropped spores ; bacterial inoculation by needle puncture, and by absorption ; the manufacture and application of various spraying mixtures ; seed treatment with chemicals ; the Jensen oat treatment ; and field inoculations and studies. The student works under direct supervision in the laboratory or field and definite reports are required.

The student is prepared for this course by thorough work in general botany, supplemented by courses in physiology, ecology and mycology, and by training in other college departments. A special effort is made to have the general botanical training as thorough as possible ; and the young man who early makes the shallow plea for practical botany is, if possible, made to feel that one can no more be the best practical botanist without being at the same time a theoretical botanist than he can be the best theoretical botanist without being a practical botanist. The effort, in short, is to make the student see that pure and applied botanical science are so correlated that each is at every point dependent upon the other. In fact we have, even in isolated colleges of liberal arts, become so influenced by the call for vocational training that, while such technical courses as the one discussed in this paper may be admitted and given wherever there is sufficient demand and proper facilities, the college teacher needs constantly to emphasize the necessity for the best possible preparation in pure science.

Fourteen young men took the course last year, a larger number than we had expected or desired. So far as demand is concerned, the course is amply justified. The work can be greatly improved with experience and by a continuation of friendly suggestions from plant pathologists ; but the young men in the course showed excellent interest and enthusiasm, even in the first presentation of this work.

In closing, thanks are due to Mr. Roy C. Faulwetter who, as student assistant in preparing and in giving the course, has rendered very efficient service and lightened many burdens which would otherwise have rendered the preparation of laboratory experiments, laboratory manual and lecture outlines impossible.

MIAMI UNIVERSITY,
OXFORD, OHIO

A FURTHER STUDY OF SOME GLOEOSPORIUMS AND THEIR RELATION TO A SWEET PEA DISEASE

J. J. TAUBENHAUS

(WITH PLATE XVI AND ONE TEXT FIGURE)

This paper presents the results of a series of experiments which support the following points developed in a previous article :[1]

1. That the anthracnose disease of the sweet pea is due to the same organism, *Glomerella rufomaculans* (Berk.) Spauld & von Sch., that causes the bitter rot of the apple.

2. That *Gloeosporium officinale* E. & E., *Gloeosporium gallarum* Ch. Rich., and *Gloeosporium* sp. from May-apple fruit are also the same as *Glomerella rufomaculans*, since they are all able to produce the typical anthracnose disease of the sweet pea and the bitter rot of the pear.

The first year (1910) the inoculations were carried on at a time when both the apples and the pears were almost mature, and ripe fruits being a more favorable medium, since they are physiologically less active than young ones, it was felt advisable to start the present inoculations of the apples and pears in the orchard at a much earlier date, this time using more organisms. The inoculations were begun in 1911 when the fruits were the size of a grape, and were repeated at various stages of their development. The Kieffer pear and the Rubicon and Paradise Sweet apples were selected for this purpose. The inoculations were made by means of punctures through the cuticle. For each organism a different sterilized needle was used. Natural infection[2] of the inoculated fruits was prevented by means of heavy paper bags which were tied to the limbs to which the inoculated fruits were attached. Any inoculated fruits which happened to drop off fell into the bags and were retained there. In every case where infection occurred it first appeared at the point of inoculation. For each organism eight fruits were used as checks. These were punctured with a sterile needle and covered with paper bags, and in all cases remained healthy. Investigations were also carried on with the sweet pea, specimens of which were grown in the laboratory from carefully selected and sterilized seeds grown in sterilized pots and soil. Checks were also used, fifty seedlings for each organism, and these in all cases remained healthy, although they were punctured with a sterilized

[1] Taubenhaus, J. J. A study of some Gloeosporiums and their relation to a sweet pea disease. Phytopathology I : 196–202. 1911.

[2] Owing to the dry summer of 1911, not a single case of bitter rot was found in the orchard. On the other hand, black rot (*Sphaeropsis malorum* Peck) was very prevalent.

TABLE I, SWEET PEA

Species of fungus	Methods of inoculation	Date of inoculation	Results from inoculation
Glomerella rufomaculans from apple	Atomizer	June 16	June 28, 99% infection
Glom. rufomaculans from sweet pea	"	May 21	June 2, 92% "
"	"	May 25	June 6, 90% "
"	"	June 16	June 21, 95% "
"	"	June 29	July 11, 41% "
"	"	July 27	Aug. 10, 80% "
"	"	Nov. 1	Nov. 10, 100% "
Gloeosporium gallarum	"	June 16	June 28, 91% "
Gloe. piperatum	"	May 25	June 12, no "
"	Puncture	"	June 12, 2% "
"	"	June 29	July 6, 41% "
"	Atomizer	June 4	June 21, no "
"[1]	"	July 27	Aug. 9, 50% "
Glom. gossypii	"	May 21	June 2, 82% "
"	"	May 25	June 12, 60% "
"	"	June 21	July 7, 100% "
"	"	June 29	July 7, 80% "
Gloe. diospyri	"	Oct. 28	Nov. 10, 82% "
Colletotrihum phomoides	"	Oct. 28	Nov. 10, 80% "
C. nigrum	"	Oct. 28	Nov. 10, 92% "
C. gloeosporioides	"	July 3	July 14, no "
"	Puncture	Aug. 1	Aug 10, " "
Glom rufomaculans from fig	"	May 21	June 2, " "
"	Atomizer	May 21	June 2, " "
Gloesporium from Populus deltoides	"	June 29	July 11, " "
"	Puncture	May 25	June 12, " "
C. lindemuthianum	"	Aug. 1	Aug. 10, " "
"	Atomizer	Aug. 1	Aug 10, " "
Gloe. musarum	"	July 27	Aug. 10, " "
"	Puncture	July 27	Aug. 10, " "
C. lagenarium	"	Nov. 1	Nov. 10, " "
"	Atomizer	Nov. 1	Nov. 10, " "

needle. Only spores from pure cultures were used for the inoculations. The results obtained are given in Table I.

From Table I it will be seen that *Glom. rufomaculans* from apple and sweet pea, *Gloe. gallarum* Ch. Rich., *Glom. gossypii* (South) Edg., *Gloe. diospyri* E. & E., *Colletotrichum phomoides* (Sacc.) Chest., and *C. nigrum* Ell. & Halst. produce the typical anthracnose disease on the sweet pea (Pl. XVI, Figs. 5, 6, 17, 18, 19) and the symptoms produced by all the above organisms were identical with those produced by the original Gloeosporium isolated from diseased sweet pea plants in the field (Pl. XVI, Figs. 1 and 3). Many more inoculations than are indicated in Table I were made with the above named organisms on the sweet pea. They were omitted from the table, since the results

[1] Gloeosporium piperatum reisolated from sweet pea seedlings infected by puncture inoculation.

obtained were similar to those given above. The data in Table I further show that *Gloe. piperatum* E. &. E. failed to infect the sweet pea by atomizer inoculation, while infection by puncture inoculation was fairly successful. When this organism was reisolated from seedlings infected by puncture, it regained its virulence, and then became able to infect sweet pea seedlings by atomizer inoculation. *Glom. rufomaculans* from the fig failed to infect the sweet pea, and, as will be seen later, it also failed to infect apples and pears on the tree. Edgerton[1] states that the above two organisms readily lose their virulence when grown for some time on artificial media. The *Gloeosporium* sp. from *Populus deltoides*, *Colletotrichum lindemuthianum* (Sacc. & Magn.) B. & C., and *Gloe. musarum* Cke. & Mass. failed to to infect the sweet pea after repeated trials both by puncture and atomizer inoculations. In comparing Tables I and II we see that the organisms which infect the sweet pea also infect the apple, with the exception, however, of *Gloe. gossypii*, which readily infects the sweet pea by atomizer inoculation, but always fails to infect apples on the tree (Pl. XVI, Figs. 5 and 6).

The data in Table II show that none of the organisms used could infect the Rubicon apple on the tree when the fruits were about the size of a large grape. Later, however, by June 26, the first positive infection was obtained with *Glom. rufomaculans* from the apple. At this same date all the other organisms used failed to infect. On July 15 the same condition prevailed. By August 19, typical bitter rot infections were obtained with *Glom. rufomaculans* from the apple and sweet pea, *Gloe. officinale*, *Gloe. gallarum*, *Gloeosporium* sp. from May-apple fruit, and *Gloe. piperatum*. Negative results were obtained with *Glom. rufomaculans* from fig, *Glom. gossypii*, *Gloeosporium* sp. from *Populus deltoides*, *Colletotrichum lindemuthianum* and *Gloe. musarum*. Further inoculations were made on the Rubicon apple until September when the fruits were already ripe and the results were the same as mentioned above.

Field inoculations were tried on the Paradise Sweet apple with the organisms mentioned in Table II and with like intervals of time between them. The results obtained were the same as with the Rubicon except that positive infection with *Glom. rufomaculans* from apple was not obtained before Sept. 7. Inasmuch as the Paradise Sweet is a late variety this indicates the greater resistance of late over early varieties.

[1] Edgerton, C. W. Diseases of the fig tree and fruit. La. Agr. Exp. Sta. Bul. 126:7. 1911.

TABLE II. RUBICON APPLE

Species of fungus	No. fruits inoculated	Dates of inoculation	Results from inoculation
12 species named below	6 fruits for each organism	June 2	June 12, all healthy
Same as above	"	June 6	June 21, all healthy
Glomerella rufomaculans from apple	8	June 14	June 26, all typical bitter rot
11 other species named below	8	"	" all healthy
Glom. rufomaculans from apple	8	June 27	June 15, all typical bitter rot
Glom. rufomaculans from sweet pea	10	"	" 8 fruits show small spots 2 fruits are healthy
Gloeosporium from May-apple	10	"	" same as above
9 other species named below	10 fruits for each organism	"	" all healthy
Glom. rufomaculans from apple	8	July 7	Aug. 2, all typical bitter rot
Glom. rufomaculans from sweet pea	10	"	" "
Gloeosporium officinale	10	"	" "
Gloe. gallarum	10	"	" "
Gloeosporium sp. from May-apple	10	"	" "
Colletotrichum phomoides	10	"	" "
Gloe. piperatum	10	"	" all very small spots, but typical bitter rot
C. gloeosporioides	10	"	" very small spots but not typical bitter rot
Glom. gossypii	10	"	" same as above
Gloeosporium sp. from Populus deltoides	10	"	" all healthy
C. lindemuthianum	10	"	" "
C. lagenarium	10	"	" "

Field inoculations were also carried on with Kieffer pear fruits. The latter being a variety which ripens very late, positive infection on the fruit with six of the organisms could not be obtained earlier than Oct. 6. The above fungi which produce the anthracnose disease of the sweet pea and the bitter rot on the apple also produce typical bitter rot on the Kieffer pear.

The foregoing experiments have conclusively proved that the anthracnose disease of the sweet pea is identical with *Glom. rufomaculans* which produces the bitter rot of the apple. Moreover, the six organisms above mentioned, which were previously considered as

distinct species, are now shown through the above experiments to be identical with *Glom. rufomaculans*, since they readily produce the typical anthracnose disease on the sweet pea and the bitter rot of the apple and the pear fruits on the tree. The experiments further indicate the saprophytic nature of *Glom. rufomaculans*[1] since no infection could be obtained on very young apples or Kieffer pear fruits on the tree. In the Delaware bulletin just referred to an explanation is given of the causes of the difference in resistance between different varieties of the same fruits and between young and older fruits of the same variety. If we look for an explanation as to why *Glom. gossypii* infects sweet peas and fails to infect apples and pears on the tree but readily infects the same fruits when they are picked and placed in moist chambers in the laboratory, we are brought to the following theory:—It seems that the *Glom. gossypii* at one time was identical with *Glom. rufomaculans*, but that through long association with the cotton plant it has become so modified in its habits as to make it a physiological species capable of infecting the sweet pea and possibly other hosts, but having lost the power to infect the apple. From this it would seem that it is the cell contents of the host which may in some cases modify the physiological habits of an organism. To refute the above statement it could not be argued that the sweet pea can be infected by any species of Gloeosporium. This is not the case, since experiments have proven that only the organisms which infect the apple can also infect the sweet pea.

The writer hopes to continue experiments along these same lines with the object of finding out whether certain other supposedly different species of the Glomerella type are not one and the same.

MODE OF INFECTION AND PERIOD OF INCUBATION

The anthracnose of the sweet pea is mainly a disease of the tender parts of the plant. Infection starts at the tips and the fungus works downwards (Pl. XVI, Fig. 1), invading both stem and leaflets until it reaches a node on the older parts of the stem, where it is stopped in its course. It is not infrequent, however, to find whole branches dying, and sometimes the entire plant is involved. In such cases it has been found that the plant is suffering from insect attacks, either by plant aphids (*Aphis* sp.), or more especially the red spider, (*Tetranychus bimaculatus* Haw.). These help the fungus both by weakening the host plant and by distributing the spores over its sur-

[1] Cook, M. T. and Taubenhaus, J. J. The relation of parasitic fungi to the contents of the cells of the host plants. Del. Agr. Exp. Sta. Bul. 94. (In press.)

face. The spores when germinating have no difficulty in penetrating the oldest parts of the host if it has been punctured by these insects. This explains why the plants suffer most during the hot dry weather, since at that time the aphids are most abundant. Infection often begins with the blossoms at the junction between the flower and the peduncle (Pl. XVI, Fig. 3), in which case the blossom shrivels. The pods, also, even those which are nearly ripe, are often seen to be badly affected. Here, too, the aphids will be found to have opened the way for the fungus. These symptoms are observed in the field when infection takes place naturally, or in the laboratory where the plants are artificially inoculated with *Glom. rufomaculans* from the sweet pea. The same mode of infection and the same symptoms are observed with the other organisms which are capable of infecting the sweet pea (Pl. XVI, Figs. 5, 6, 17, 18, 19).

The spores of *Glom. rufomaculans* from the sweet pea usually germinate in from six to twenty-four hours, according to the amount of moisture in the atmosphere. The germ tubes enter the host by breaking through the epidermal cells of either leaf or stem (Fig. 1). In case the spore lodges on a stomate, the germ tube grows away and avoids entrance. The exchange of gases which takes place at the stomates renders them toxic and prohibits the entrance of the germ tube, which often breaks through the epidermal cells as soon as the spore germinates. At other times the spore germinates by sending out a short germ tube which forms an appressorium which attaches to the epidermal wall. This appressorium is then seen to germinate, its germ tube breaking through the epidermal cell (Fig. 1).

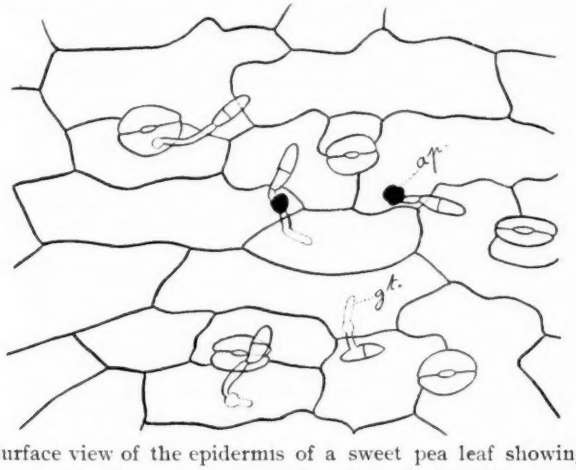

FIG. 1. Surface view of the epidermis of a sweet pea leaf showing the germ tubes of the sweet pea Gloeosporium breaking through the cells : *ap.*, appressoria; *gt*, germ tube.

The period of incubation varies from three to five days according to the amount of moisture in the atmosphere. The acervuli appear within five days after wilting begins unless the weather be dry, when they may not appear until eight to ten days after infection. In the field the sweet pea anthracnose is at its height during July and August. This is also the time when the bitter rot of the apple makes its appearance in the orchard. It is thus easy to understand how readily the natural cross inoculation can be effected.

SUMMARY

1. It has been proved by many cross inoculations on the apple and the sweet pea that the anthracnose disease of the latter is due to the same organism, *Glomerella rufomaculans* (Berk.) Spauld. & von Sch., as causes the bitter rot of the apple.

2. *Gloeosporium gallarum* Ch. Rich., *G. diospyri* E. & E., *G. officinale* E. & E., *Gloeosporium* sp. from May-apple fruit, *Colletotrichum nigrum* E. & H. and *C. phomoides* (Sacc.) Chest. appear to be identical with *Glom. rufomaculans* (Berk.) Spauld. & von Sch., since they can produce the typical bitter rot of the apple on the tree and the anthracnose disease of the sweet pea.

3. *Glomerella gossypii* (South) Edg. seems to be a physiological species of *Glom. rufomaculans*, since it readily infects the sweet pea. It cannot infect the apple on the tree unless the fruit is mature or is picked from the tree and brought indoors.

4. *Colletotrichum gloeosporioides* Penz., *Gloeosporium* sp. from *Populus deltoides*, *G. musarum* Cke. and *C. lagenarium* (Pass) E. & H. seem to be distinct species, since they are unable to infect the apple or the sweet pea even by puncture inoculation.

5. The great number of our species of Gloeosporium and Colletotrichum would no doubt be reduced if we were to put them to the test of cross inoculations.

6. Infection with *Glomerella rufomaculans* on the sweet pea is affected when the germ tube of the germinated spore breaks through the epidermal cells of the tender leaves or stems.

7. The period of incubation of *Glom. rufomaculans* on the sweet pea ranges from three to five days according to the amount of moisture in the atmosphere.

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DESCRIPTION OF PLATE XVI

Fig. 1. Sweet pea plant naturally infected in the garden with *Glomerella rufomaculans*.
Fig. 2. Healthy blossom of the sweet pea.
Fig. 3. Sweet pea blossom naturally infected with *Glomerella rufomaculans*.
Fig. 4. Check seedlings (healthy) of sweet pea.
Fig. 5. Sweet pea seedlings artifically infected with *Glomerella gossypii*.
Fig. 7. Check apple (healthy).
Fig. 8. Apple artificially infected on the tree with *Glomerella rufomaculans* from the apple.
Fig. 9. Apple artificially infected on the tree with *Glomerella rufomaculans* from the sweet pea.
Fig. 10. Apple artificially infected on the tree with *Gloeosporium gallarum*.
Fig. 11. Apple artificially infected on the tree with *Gloeosporium* sp. from May-apple fruit.
Fig. 12. Apple artificially infected on the tree with *Gloeosporium officinale*.
Fig. 13. Apple artificially infected on the tree with *Colletotrichum phomoides*.
Fig. 14. Apple artificially infected on the tree with *Colletotrichum nigrum*.
Fig. 15. Apple artificially infected on the tree with *Gloeosporium diospyri*.
Fig. 16. Check seedlings of sweet pea.
Fig. 17. Sweet pea seedlings artificially infected with *Gloeosporium diospyri*.
Fig. 18. Sweet pea seedlings artificially infected with *Colletotrichum phomoides*.
Fig. 19. Sweet pea seedlings artificially infected with *Gloeosporium gallarum*.

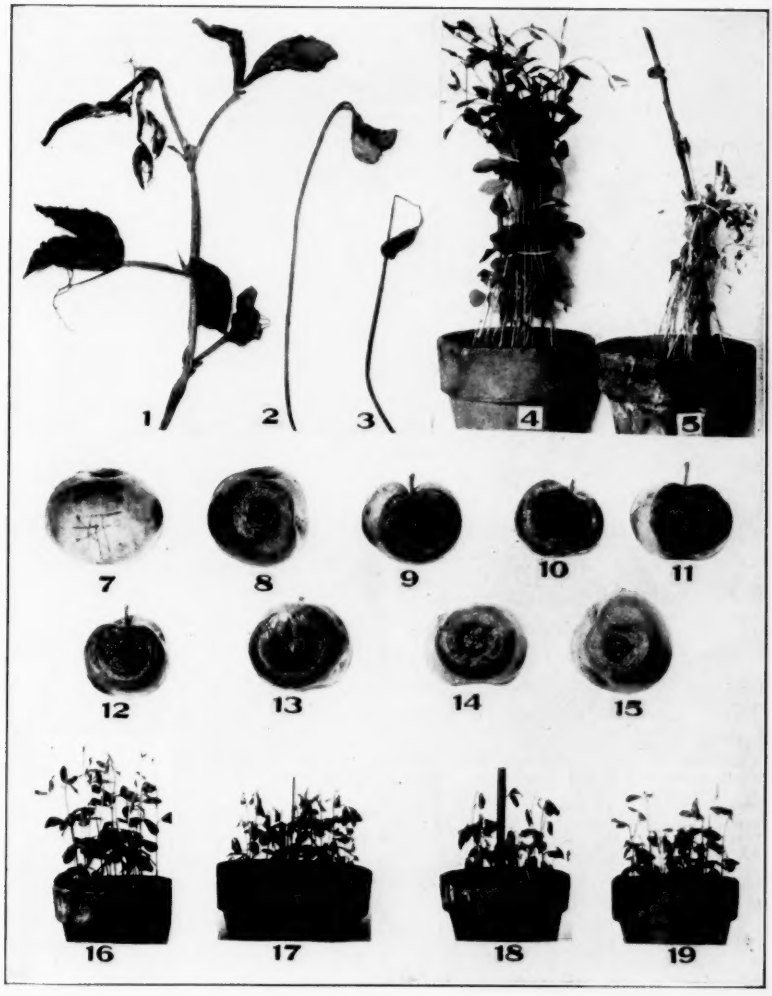

PLATE XVI—**Taubenhaus on Gloeosporium**

THE TREND OF INVESTIGATION IN PLANT PATHOLOGY

H. A. HARDING

The earlier workers in plant pathology were such mainly by accident since at first there was necessarily no recognized field of plant pathology. Smith has well characterized them as pioneers, poorly equipped for the problems which they were to encounter, traveling without chart or compass. All honor is due to their heroic labor in the face of unfavorable environment. We should have much charity for the individuals who labored under many handicaps but we should not thoughtlessly accept their conclusions when based on erroneous data or their generalizations when resting upon false analogies. While it is true that the pioneers reasoned that there could be no bacterial plant disease because the reaction of plant juices is acid and bacteria grow only on alkaline media, and made some other mistakes, the greatest sins which they committed were those of omission. It is mainly these sins of omission which the present trend of plant pathology is tending to correct.

The earlier work was done by botanists who focused their attention upon the host. Many of their morphological and histological studies of the host might well serve now as models for work on that phase of the subject. We have added little to our knowledge of the histology of legume nodules since Beijerinck's paper in 1888. With the recognition of the interesting character and the economic importance of the parasite there came those who knew little or cared little concerning the morphology of the host but whose interest was centered upon the parasite. To find a new parasite and write after its name their own mystic symbols was the height of their ambition. As a result plant pathology became largely parasitology and the host was all but forgotten.

The economic importance of the host gradually returned it to scientific notice and to conserve the host there was developed "spray-ology" in which the aim seemed to be to find a mixture with a new color or a new smell which might be applied several times per year with great profit to all concerned. This has developed to such an extent that at least in the Eastern horticultural regions the vernal landscape now rivals the famous coat of Joseph and the perfume laden air bears the added aroma of Hades.

Underneath this confused mass, much of which is superficial and temporary, there has been steadily building the foundation of plant

pathology. The period when specialization in research ran riot and it was fashionable to consider only a minute angle of the problem in hand is passing. The conviction is growing that we can not study a problem intelligently without considering it in relation to as much of its environment as our own equipment and the development of science will permit.

Along with this broader and deeper outlook into the relations of the parasite and the host there has arisen the study of the avenues through which the parasite enters, of the biochemistry of the attack, of the physiological and morphological responses of the host. In short we are steadily and rapidly adopting in plant pathology the point of view which rules in the older and more fully developed sister science—animal pathology. According to this view pathology is the science of disease and is concerned almost exclusively with the host. The problem of the pathologist is first to understand the cause and the nature and extent of the abnormalities of the host. When these are understood he may proceed intelligently to devise means for their relief or future prevention.

The ideas which we have already adopted from animal pathology are many and important. On the basis of microscopical examination and without any inoculation experiments Beijerinck asserted the causal relation of bacteria to the formation of legume nodules and many later workers have made just as reckless and far less accurate guesses. Koch's canons came to us from animal pathology and today I think no plant pathologist would consider the causal relation of a plant parasite satisfactorily proved until these canons had been fulfilled.

The earlier workers considered that the possession of pathogenicity, in itself, was a sufficient characterization of the causal organism and gave slight attention to further descriptions of the parasite. Many years ago bacteriologists were taught by their experiences in animal pathology that pathogenicity was a physiological function which could be lost and regained under a variety of conditions. Accordingly they made a fairly extensive study of the physiological functions of their organisms as influenced by environment. As the result of these studies the bacteriologists now have a moderately satisfactory basis for description and classification which takes account of both morphology and physiology.

The students of fungi have been slower to develop and organize their physiological studies, possibly because their available morphology was more satisfying. However the fact is recognized that pathogenicity is more a matter of physiology than of morphology. Within the

recognized species of fungi, based as they are upon morphology, there may be, and often are, a number of physiological races. With the growth of this conception of physiological races the physiological side of fungus study is certain to receive more attention.

If there is a doubt in the mind of any one as to the objective point toward which the development of plant pathology is tending let him read the second volume of Erwin F. Smith's "Bacteria in Relation to Plant Diseases." There he will find 200 pages devoted to the presentation of the science of plant pathology and its form of presentation is almost identical with that current in animal pathology. Whenever this is possible the terminology of animal pathology is applied to the corresponding tissue changes in plants. It is entirely natural that this growing correlation between animal and plant pathology should be most evident in the treatment of bacterial diseases since many of the bacteriologists, in the nature of the case, have come into closer touch with animal pathology than have the students of fungi.

One of the regrettable features in connection with this newer development of our science is the fact that those of us who were victims of the craze for specialization in training, which characterized the past two decades, find ourselves, like the original pioneers, poorly equipped for doing work which shall attain the desired standard of excellence. Notwithstanding these limitations in our original training, if we are to produce results which shall command the respect of our colleagues and the commendation of our successors we must enlarge our horizon in every possible way. Much can be accomplished by applying ourselves to the study of related branches of knowledge but with the older workers the burden of other duties makes this difficult.

In the case of two or more men who are temperamentally fitted to work together great gains result from cooperative work. With many problems cooperation furnishes the only means by which their solution is possible. While scientific cooperation has its dangers and limitations it will undoubtedly be responsible for some of the most striking advances of the coming generation.

The present trend of investigation in plant pathology may be characterized as an effort to follow animal pathology in recognizing the host as the focal point of study and to consider the relation of the host to its environment in a comprehensive way. The limiting factor in this effort is the narrow training of the workers. While this can be partially overcome by personal studies it can be removed best by cooperation.

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NOTES ON THREE SPECIES OF RUSTS ON ANDROPOGON

WILLIAM H. LONG

During the spring of 1908, at Denton, Texas, the author found aecia on *Oxalis corniculata* L. associated with a Puccinia on *Andropogon furcatus* Muhl. In 1910 and 1911 aecia on *Viola sagittata* L. near Clarendon, Virginia, were found associated with *Puccinia ellisiana* Thuem. on *Andropogon virginicus* L.; while in 1912 aecia on *Viola primulifolia* L. were collected intermixed with the dead leaves of *Andropogon virginicus* L. bearing teliospores of *Uromyces andropogonis* Tracy. Aecia were also found during 1912 on *Viola sagittata* L., *V. fimbriatula* Sw. and *V. papilionacea* Pursh., in each case associated with *Puccinia ellisiana* Thuem. on *Andropogon virginicus* L.

Some of the dead leaves bearing the teliospores of the Puccinia found at Denton, Texas, associated with the aecia on *Oxalis corniculata* L. were carried to the laboratory and inoculations made April 1 on the Oxalis under control conditions. April 7 pallid spots appeared and April 12 aecia were beginning to show, but unfortunately the Oxalis was infested with flea beetles, which the writer attempted to destroy by fumigating with chloroform. The beetles were killed, so was the plant. No mature aecia were therefore obtained, but the experiment had progressed far enough to prove that the aecia on Oxalis found in the field was the alternate stage of this Puccinia on the Andropogon.

In 1910 some leaves from the Andropogon infested with *Puccinia ellisiana* at Clarendon, Virginia, were collected and successful inoculations made in the greenhouse on two species of violets, *Viola sagittata* L. and *V. papilionacea* Pursh. In 1911 similar results were obtained on these two violets for the Puccinia, while cultures were tried with *Uromyces andropogonis* Tracy, but were successful only on *Viola primulifolia* L. This spring (1912) both the Uromyces and the Puccinia were sown on seven species of violets under control conditions in the greenhouse. Parallel cultures, made as near the same dates as possible for a given species of violet, were attempted for both *Uromyces andropogonis* and *Puccinia ellisiana*. Check or control pots of each species of violets were kept in every instance.

The following shows in detail the results obtained from each set of cultures made with the Uromyces and with the Puccinia. The teliosporic material for the Uromyces was collected by the writer at Arlington, Virginia, April 14, 1912, and the Puccinia was collected at Vinson Station, Virginia, March 24, 1912, and at Cherrydale, Virginia, April 12, 1912, all having as the common host *Andropogon virginicus*

L., and much of it after some germination of the teliospores had occurred.

I. *Puccinia ellisiana* Thuem. on *Andropogon virginicus* L. was sown on the following species of violets :

(1). *Viola pedata* L., April 10, and again on April 15, with no results.

(2). *Viola fimbriatula* Sw., April 19, with infection showing May 6 on all the plants inoculated and with aecia on May 9.

(3). *Viola primulifolia* L., April 19, with no results.

(4). *Viola hirsutula* Brainerd, April 22 ; pycnia May 8, and one group of aecia just showing May 16 on one of the two plants inoculated. Slugs destroyed this before the maturity of the aecia.

(5). *Viola sagittata* L., April 15, with no results, the plants used being too mature for infection. This species was successfully inoculated with this rust in 1910 and again in 1911.

(6). *Viola papilionacea* Pursh, April 2, pycnia, April 11 and aecia, April 17, infection strong and abundant on five out of six pots of violets. This host as noted above was successfully inoculated with this Puccinia in 1910 and in 1911.

(7). *Viola cucullata* Ait., April 15, with no results.

II. *Uromyces andropogonis* Tracy on *Andropogon virginicus* L. was sown on the same species of violets as the Puccinia and in most instances on the same day for any given species.

(1). *Viola pedata* L., April 15, with no results.

(2). *Viola fimbriatula* Sw., April 19, with no results.

(3). *Viola primulifolia* L., April 19, pycnia, April 30, aecia, May 8. The infection was so severe that the leaves were seriously injured, some dying before the maturity of the aecia ; every plant (3 pots) and every leaf on these was infected. Similar results were obtained in 1911 on this host.

(4). *Viola hirsutula* Brainerd, April 22, with no results.

(5). *Viola sagittata* L., April 15, with no results.

(6). *Viola papilionacea* Pursh, April 15, with no results.

(7). *Viola cucullata* Ait., April 15, pycnia, April 27, aecia, May 6. The violets in two out of three pots inoculated were infected, but the infection was weak, the affected areas small, and the hypertrophy slight. The infection was general on one plant, as some six or eight leaves, including petioles, were attacked. The aecia and spores, however, were characteristic of *Uromyces andropogonis* Tracy.

III. *Puccinia ellisiana* Thuem., I on *Viola sagittata* L. and *V. papilionacea* Pursh. was sown on *Andropogon virginicus* L. May 10,

uredinia appeared May 22 in abundance on ten or fifteen leaves in each pot. The aeciospores from both species of violets infected with equal vigor the grass leaves.

IV. *Uromyces andropogonis* Tracy, I on *Viola primulifolia* L. was sown on *Andropogon virginicus* L. May 10. One or two uredinia appeared June 5.

A study of the results shows that of the species of violets inoculated with *Puccinia ellisiana*, three, viz., *Viola primulifolia*, *V. pedata*, and *V. cucullata*, gave negative results, while for *Uromyces andropogonis* only two species, *V. primulifolia* and *V. cucullata*, were infected, leaving *Viola pedata* not infected by either rust. This may not hold true for future tests, since this violet is reported to harbor an aecium similar to the aecia produced by these two rusts, and, by taking it at just the right stage of development, infection may occur. Negative results with these two Andropogon rusts are of little value, since the infection of a given species of violet one season and the failure to infect the same species the next season may and does occur. There seems to be a period of susceptibility in the development of the violet, before and after which infection is not possible, or at least is rare. This has been clearly shown in the three seasons' cultural work of the writer with *Puccinia ellisiana*; also in the successful attempt of Dr. Sheldon to infect *Viola sororia* Willd. with *Uromyces andropogonis* (Torreya **9**: 55. 1909 and **10**: 9. 1910); and the failure of Dr. Arthur to secure infection on violets with the same rust one season (Mycol. **1**: 232. 1909) and the partial success—pycnia only being obtained—the next year (Mycol. **2**: 228. 1910). Added to this are the writer's success during three years with cultures of *Puccinia ellisiana* on several species of violets, here reported for the first time, and Dr. Arthur's failure to secure infection with the same rust when sown on violets (Mycol. **2**: 220. 1910). But when, in parallel cultures made on the same host, same date, under identical conditions, the Puccinia infects and the Uromyces does not, and vice versa, which is exactly what occurred in this year's cultural tests, it would seem to indicate that the two rusts have different species of violets for their aecial host. A similar condition seems to exist in the field where *Viola sagittata*, *V. fimbriatula* and *V. papilionacea* are commonly found infected with the Puccinia, and no Uromyces present, while *Viola primulifolia*, the common and only host found in the field for the Uromyces in the localities examined, is not attacked by the Puccinia.

In a moist meadow at Arlington, Va., the writer found *Uromyces andropogonis* abundant on *Viola primulifolia*, but none on two other

species of violets (*V. sagittata* and *V. papilionacea*) growing intermixed with the infected *V. primulifolia*. However, the writer has not yet found *V. pedata* associated with infected *V. primulifolia*, so there are no field data from which to judge as to the susceptibility of *V. pedata* to the Uromyces. Through the kindness of Dr. Sheldon, the writer was able to examine fresh material of the violet infected with *Uromyces andropogonis* from his locality. It is neither of the species here noted as hosts for this rust, but is *Viola sororia* Willd.

It is not impossible that *V. pedata* may be the aecial host for *Uromyces andropogonis*, but there are neither field nor laboratory data known to the writer that prove the relationship between the aecium reported on this host and *Uromyces andropogonis*. It may be *Uromyces andropogonis*, it may be *Puccinia ellisiana*, or it may be the aecium of some other rust. In three different localities in Virginia the writer has found *V. pedata* associated with other species of violets affected with the Puccinia rust, but in no case was a single plant of *V. pedata* attacked, even when standing side by side with infected plants of the other species. From these field observations, together with the laboratory results noted above, the aecium reported on *V. pedata* is probably not this Puccinia; nevertheless, further investigation may prove differently. It would seem from the evidence at hand that *Uromyces andropogonis* should not be called *Uromyces pedatata* till positive proof is advanced of the relationship of the teliosporic stage on the Andropogon to the aecium reported by Schweinitz on *V. pedata* (Trans. Am. Phil. Soc. II, **4**: 293. 1832). On the other hand, both from field data and from laboratory cultures, the aecium reported on *V. sagittata* and called by Schweinitz (Trans. Am. Phil. Soc. II. **4**: 293. 1832) *Caeoma* (*Aecidium*) *sagittatum* is undoubtedly the aecial stage of *Puccinia ellisiana* which should therefore be known as **Puccinia sagittata** (Schweinitz) n. comb. Furthermore, the brief description of this aecium given by Schweinitz agrees with the characters of *Puccinia ellisiana*, but would apply equally as well to the aecium of *Uromyces andropogonis*. If cultures should prove that both *Uromyces andropogonis* and *Puccinia ellisiana* have *V. pedata* as their aecial host, there would be no means of knowing to which rust the specimens of Schweinitz found on this host belonged.

In the report of "Cultures of Urediniae for 1910" (Mycol. **4**: 14. 1912), and again in a paper read before the meeting of the Phytopathological Society at Washington, D. C., in December, 1911, Dr. Arthur called attention to the marked similarity in the morphological characters of certain species of Puccinia and Uromyces, and questioned

the validity of the genus Uromyces except as a matter of convenience. Bearing this in mind, the writer made a special study of the aecia of *Uromyces andropogonis* and of *Puccinia ellisiana*, trying to determine all differences as well as similarities in the characters of these two rusts. The aecial stage of *Puccinia violae* was also included in the study, as it is somewhat similar. Fresh material of each species was used, and the data given are based on the observations made under these conditions.

For purpose of comparison, the salient characters of the three aecia are given. A brief description is also included from dried material of the aecium on *Oxalis corniculata*, found in Texas associated with Puccinia on *Andropogon furcatus*.

PUCCINIA ELLISIANA THUEM.

I. Aecia usually hypophyllous but often seen on petioles and scapes, occasionally on the upper surface of the leaf and then opposite a group on lower surface, seated on pallid thickened oval areas 1-6 mm. in diameter, cupulate, short, aecial cavity 121-165μ across, average for ten aecia 138μ. Peridium irregularly lacerate and somewhat reflexed, distinctly yellow under hand lens when first expanded, later becoming whitish ; peridial cells, pulvinate in side view, with the base as the outer wall of the peridium, polygonal (3-5 sides) in face view, contents of lumen yellow when seen under compound microscope. In face view the yellow centers and broad white margins of the cells are very characteristic. Peridial cells 15-19μ thick x 15-30μ long, plainly overlapping, outer wall in face view appearing finely punctate-striate with the striations more or less parallel, transversly striate in side view, 5-8μ thick ; inner wall 2-3μ thick, verruculose, aeciospores 12-17 x 16-18μ, average size for ten spores 15 x 17μ.

UROMYCES ANDROPOGONIS TRACY

I. Aecia same as in *Puccinia ellisiana*, except that the peridial walls seem to fade quicker to a dirty white. Peridial cells in size, color and sculpturing the same as those of the Puccinia ; aeciospores have the same shape, sculpturing and color as the Puccinia, while their size varies slightly for the different hosts as follows : On *Viola primulifolia* (the common host in the vicinity of Washington, D. C.), 13-16 x 14-16μ, average for ten spores 13.6 x 15μ ; for *Viola sororia*, the common host at Morgantown, West Virginia, 13-16 x 14-17.7μ, average for ten spores 15 x 17μ. For *Viola cucullata* 13-16 x 15 x 17μ, average for ten spores 14.7 x 15.9μ. A comparison of these measurements with those of Puccinia shows that they are practically the same.

PUCCINIA VIOLAE (SCHUM.) D. C.

Found on *Viola cucullata and V. sagittata.*

I. Aecia much larger than those of *Puccinia ellisiana* and *Uromyces andropogonis*, with a more even and pronounced reflexing of the peridial walls. Aecial cavity 220-286μ across, average size for ten, 252μ, or nearly twice as large as in the other two rusts under consideration. Peridial cells white under compound microscope, pulvinate in side view, very slightly overlapping, 22-32μ thick by 32-40μ long, average for six cells 26 x 35.5μ; outer walls 8-10μ thick; inner walls 3-4μ thick; outer wall in face view plainly punctate but the points not arranged in parallel lines like those of the aecia of the two Andropogon rusts; transversely striate in side view; inner wall verruculose with rather coarse irregular granules. Aeciospores subglobose, 16-20 x 22-26μ, average for ten spores 20 x 24μ, verruculose.

PUCCINIA ON OXALIS CORNICULATA

I. Aecia seated on pallid spots in circular groups of 4-10 or more, hypophyllous, cupulate, short; aecial cavity 154-220μ across, average size for ten aecia, 175μ. Peridium irregularly lacerate and (in herbarium material at hand) breaking away entirely; color when fresh not noted. Peridial cells pulvinate in side view, polygonal (3-6 sides) in face view, 15-18μ thick by 20-30μ long; outer wall 5-7μ thick, striate in side view, in face view appearing finely punctate-striate, with the striations more or less parallel, like the other two Andropogon rusts under discussion; inner wall about 3μ thick, minutely verrucose. Aeciospores subglobose, verruculose, 16-19 x 16-23μ, average for ten spores 17 x 18μ.

This aecium is very similar in all its characters to the Andropogon rusts on violets. However, the aecia are somewhat larger, and the peridial cells flatter, but still distinctly pulvinate.

The most salient differences between the aecia of *Puccinia violae* and the Andropogon rusts which have their aecia on violets are: larger aecia, nearly twice the size of the other rusts; white peridial cells under high power of compound microscope; thicker and longer peridial cells, with very slight overlapping and coarser markings on both walls; absence of the parallel ridge-like markings on outer wall (face view); and larger spores. The overlapping of the peridial cells of the Andropogon rusts is so pronounced that when the aecia open they tear very irregularly, the upper part often breaking off entirely, leaving a shallow cup with eroded edges, in place of the plainly recurved walls of the peridia, as seen in *Puccinia violae.*

A careful comparison of the aecial, uredinial, and telial characters of *Puccinia ellisiana* and of *Uromyces andropogonis* shows no fixed morphological differences, so far as the writer has been able to discover, except the teliospores of the former are two-celled, while those of the latter are one-celled. However, both from laboratory cultures and field data, the two rusts seem to have their aecial stage on different species of violets. Here the common aecial hosts for the Puccinia are *Viola sagittata*, *V. fimbriatula*, and *V. papilionacea* ; for the Uromyces, *Viola primulifolia*, and at Morgantown, West Virginia, *Viola sororia*. However, more extended investigation may show that both species occupy the same aecial host in some instances. The two species have many characteristics in common, as has been noted by other writers. One noticeable feature in both is the paucity of urediniospores and the briefness of their duration. This peculiarity has produced a marked effect on the distribution of these rusts on the gramineous host, viz., neither rust is ever, or very rarely, found any distance from bunches of violets ; in every instance coming under the writer's notice, the stools of grass bearing the uredinia and telia had violets growing at their base, or within two feet of them. The main function of the urediniospores seems in this case to be the distribution of the rust on the later leaves of the same stool of grass, rather than to distant bunches.

The taxonomic position of these two rusts and their relationship to each other must be determined by further study and cultures. The writer in 1910 sent some of this same Puccinia (apparently the same) to Dr. Arthur, who successfully produced aecia on Penstemon from it, and it was referred by him to *Puccinia andropogonis* Schw. (Mycol. **4**: 17. 1910). Also in a letter he stated that the Andropogon leaves sent him apparently had two species on them, viz., *Puccinia andropogonis* and *Puccinia ellisiana*. If such was the case then his results were to be expected, but the writer has never found any species of Penstemon in the vicinity of the Andropogon plants infected with this rust, although he has carefully looked for them for three years ; while on the other hand, he has always found the Puccinia associated with the aecia on the violets, from which it would seem that either the Penstemon rust has so far escaped observation, or that this Puccinia has its alternate stage on both violets and penstemons. Several collections of Puccinia on Andropogon were sent from Denton, Texas, by the writer to Dr. Arthur, and identified by him as *Puccinia andropogonis* Schw., with its aecium on penstemon, yet in some eight years careful study of the rusts in that region the writer has never found a

single Penstemon bearing aecia, while *Oxalis corniculata* was collected with the aecia of an Andropogon rust, and cultures as noted above were made from the rusted Andropogon leaves. To date, four families of plants are known to harbor aecia of the Andropogon rusts, to wit: *Scrophulariaceae*, *Violaceae*, *Santalaceae*, and *Oxalidaceae*. In view of the above facts, it would seem that the entire group of Andropogon rusts needs a thorough study especially by cultural methods.

The writer is indebted to Drs. J. C. Arthur and F. D. Kern for the determination of the teliosporic material used in the cultures, to Dr. A. S. Hitchcock for identifying the graminaceous host, and to Mr. Ivar Tidestrom of the U. S. Department of Agriculture, and to Dr. Ezra Brainerd for the identification of aecial hosts.

OFFICE OF INVESTIGATIONS IN FOREST PATHOLOGY,
BUREAU OF PLANT INDUSTRY,
WASHINGTON, D. C.

FIELD LABORATORY EQUIPMENT

DONALD REDDICK

When the writer began his grape disease investigations in 1907 he was confronted with the necessity of moving away from the laboratories of the University to a region where grapes are grown. It was thought that much time and expense might be saved by moving equipment for laboratory work to the vineyards. A greater consideration, in the writer's opinion, is the fact that, properly equipped, one may pursue without interruption a specific problem and feel sure that he will be on the ground when important phases in the yearly cycle of a disease producing organism are occurring. Thus the writer, attacking his first strictly pathological problem, found first how to quickly obtain pure cultures of the grape black rot organism on a Sunday and had the pleasure of observing and following the hourly development of black rot lesions on the fruit on Independence day.

In equipping a laboratory for field investigations it is obvious that certain pieces of apparatus may be dispensed with as for example, a paraffin oven or a microtome, while in other cases less cumbersome, less expensive or more convenient apparatus must be substituted for the usual equipment.

The original list of equipment used in 1907, after receipt of many express and mail parcels of forgotten articles, has been modified from year to year, but the present season 17 laboratories in various parts of New York State are equipped with the following articles together with such special apparatus, reagents, machinery and so forth, as may be called for in the proper execution of the special work in hand.

Experiment Stations and Agricultural Colleges are not often located conveniently for pathological work, nor would it be possible, even if it were desirable, so to locate them in a state having such a diversified agriculture as New York. The field laboratory is a portable affair and might be transported on a day's notice to a distant locality.

Two large boxes may be found which will contain the articles mentioned below, but boxes will usually receive better treatment at the hands of expressmen if they are smaller and not too heavy. The delay and annoyance of cartage at both ends and the necessity of using much of the apparatus for teaching and so forth in the winter, precludes freight shipments and adds a reason for taking along the least amount of apparatus necessary for good work. Needless to say all apparatus must be carefully packed in excelsior and all small pieces should be packed first in small pasteboard or thin wooden boxes.

The quantities of reagents, test tubes, dishes and so forth, mentioned are usually sufficient for work with fungous diseases but must be varied as occasion demands.

Glassware. 50 shell vials, 50x25 mm. with corks; 13 reagent bottles with pipettes, 30 cc; 6 narrow-mouthed bottles with rubber stoppers (250 cc.); 2 winchesters (2 liter) for alcohol and formalin; 2 bottles for fixer; 24 specimen bottles; 3 moist chambers (190x80 mm); 6 stender dishes (140x50 mm.) with ground glass lids; 6 preparation dishes (with shoulder and flat lid) assorted sizes; 6 tumblers; 6 Syracuse watch glasses; lamp chimneys (purchased as needed); 1 cylinder (graduated, 100 cc.); 1 cylinder (graduated, 10 cc); 2 flasks, 500 cc.; 6 Erlenmeyer flasks, 150 cc.; 1 funnel, fluted, 6 or 8 in.; 1 funnel, plain, small; 100 test tubes, 6 in.; 50 petri dishes; 2 hydrometers, high and low scale; 2 stirring rods; 1 thermometer (— 20–200° C.); 1 burette; 3 standardized graduated pipettes, assorted; 2 platinum needles; 1 atomizer; 1 alcohol lamp; 6 pipette droppers.

Miscellaneous apparatus and materials. 1 balance with weights; 1 steam sterilizer[1]; 1 stew pan (aluminum); 1 double boiler; 1 granite ware pail; 1 white porcelain casserole; 1 hand sprayer; 12 tin boxes for test tubes; 1 ring stand with three assorted rings; 1 burette clamp; 1 vasculum; 2 swimming cups with corks; 1 blue flame kerosene stove; 50 absorbent paper driers; 100 collecting paper folders; 1 wax pencil; 2 towels; 12 boxes matches (safety); 1 bunch string tags; 25 pot labels; filter paper (assorted sizes).

Stationery, etc. 25 letter heads and envelopes; 200 note sheets; 50 sheets drawing paper (thin); 25 folders for notes; 1 box gem clips; 1 bottle writing fluid; 1 bottle india ink; 1 eraser; 1 pencil (6H drawing); 1 pencil (2H); 1 pen holder and points; 1 box thumb tacks; 1 package rubber bands.

Microscopic outfit. 1 microscope with lenses, including oil immersion, mechanical stage desirable; 1 camera lucida; 1 ocular micrometer (determine ratio); 1 bottle immersion oil; 12 sheets lens paper; 1 bundle pith; 1 razor; 1 hone; 1 strop; 1 curved forceps; 1 straight forceps; 1 cover glass forceps; 100 slides; ½ oz. cover glasses, No. 1, 18mm.; 4 slide boxes; 1 small scissors; 2 pointed scalpels; 2 common scalpels; 3 dissecting needles; 1 box gummed labels.

[1] A cylindrical tin vessel with perforated bottom and a tight-fitting lid made to fit snugly on a granite vessel has given excellent service. The writer has often run a hundred tubes under field conditions for one hour on two successive days and obtained 100% of the tubes sterile.

Reagents. The following standard reagents to be put up in 30cc. pipette-stoppered reagent bottles: chloral hydrate; potassium hydroxide; eosin; eosin glycerine; methylene blue; iodine; acetic acid; hydrochloric acid; ammonium hydroxide; sulphuric acid; xylol; carbol fuchsin; Canada balsam, 1 tube. Cedar oil in 250cc. bottles, 4 grades, ¼ cedar oil + ¾ absolute alcohol, 50 cc.; ½ cedar oil + ½ absolute alcohol, 50 cc.; ¾ cedar oil + ¼ absolute alcohol, 50cc.; pure cedar oil, 100 cc. Alcohol: absolute, 100 cc., 95%, 2 liters.; formalin 40%, 1 liter; Gilson fixer 500 cc.; Chrom-acetic fixer 250 cc.

Miscellaneous. ½ package agar; 1 roll absorbent cotton; 10 yds. cheese cloth; gelatine.

Photographic. 1 camera, 5x7 in.[2]; 3 plate holders, 5x7 in.; 2 kits 4x5 in.; 1 tripod; 1 focusing cloth; 1 ray filter; 2 dozen plates, Cramer's isochromatic instantaneous, 5x7; 2 dozen plates, Cramer's isochromatic instantaneous, 4x5; 1 developing tray, 5x7; 1 fixing tray, 11x15; 1 ruby lamp; 24 negative envelopes, 5x7; 24 negative envelopes, 4x5; 1 pint hypo. crystals; 500 cc. developer (made up).

Weather instruments. 1 maximum and minimum thermometer with magnet and bar; 1 rain guage; 1 thermograph; 1 psychrometer; information bulletin of the weather bureau; weather maps.

CORNELL UNIVERSITY,
ITHACA, N. Y.

[2] An attempt is being made to substitute a much smaller, lighter camera with film pack, etc., but provided with an unusually good lens.

PHYTOPATHOLOGICAL NOTES[1]

Bacterial Mulberry Blight. Those readers who have kept abreast of the literature of the bacterial mulberry blight will recall that, for the organism causing this disease in the United States, I have used Boyer and Lambert's name *Bacterium mori* with my own description, *i. e.*, such a description of the organism as will enable other workers to isolate and study it. This I could very well do because while Boyer and Lambert obtained infections and tied their name to the disease, they did not add any description of the organism worth mentioning, *i. e.*, none that would not apply equally well to any other parasitic bacterium. I write this note because in literature an earlier name than *Bact. mori* has also been associated with the mulberry blight, viz., *Bacillus cubonianus* Macchiati. I could not use Macchiati's name because he tied his name definitely to a sporogenous liquefying organism, yellowish white on agar, yellowish white on gelatin becoming sulphur yellow, and growing rapidly from the beginning on potato in sinuous-margined spots, having a yellow color which becomes deeper yellow, all of which definitely separates it from *Bact. mori* as studied and defined by me, and proved by pure-culture inoculations to be decidedly pathogenic to the mulberry. We are, therefore, forced to one of two conclusions, either there are two mulberry diseases or else Macchiati's organism is a saprophyte. All the yellow organisms isolated by the writer from mulberry and tried were non-pathogenic. I do not know that any of these were Macchiati's form, but certainly until some one has shown that such an organism as he has described is truly pathogenic, the name *B. cubonianus* should not be used for any form of the mulberry blight. Should there prove to be a second mulberry blight in Italy associated with a yellow organism, Macchiati's name *Bacillus cubonianus* might be used for it, *provided it corresponds to his description.* If, on the contrary, the Italian disease should prove to be due to the polar flagellate, white, non-liquefying organism which I have described (Science, n. s., May 20, 1910, p. 792), then the name *Bact. mori* emend. Smith, should be given to it.—ERWIN F. SMITH.

Bacillus coli, a Cause of Plant Disease. Owing to the publication a month after the appearance of J. R. Johnston's preliminary note

[1] Scientific notes not exceeding two hundred words in length, and news items of special interest to plant pathologists, will be regularly printed under this caption. Manuscripts may be sent directly to the editor in charge, Haven Metcalf, 1223 Vermont Avenue, Washington, D. C.

ascribing the coconut bud-rot to *Bacillus coli*, of a note by Dr. A. W. Giampietro claiming a soft rot of the onion to be due to this organism, it might be thought that the latter reached the conclusion that *Bacillus coli* is a plant parasite independently, but such is not the case. Giampietro's work was done in my laboratory with a full knowledge of Johnston's conclusions and was not begun until Johnston's paper was completed. The onion paper represents only an imperfect piece of work, and was published without my knowledge or consent. Some months prior to its appearance in the Rivista di Patologia Vegetale Dr. Giampietro handed me substantially the same note in English for immediate publication. I told him he could not publish it until Johnston's paper was published, because the central idea in it belonged to the latter. I wrote immediately to Johnston, who was then in Porto Rico, that there were unavoidable delays in getting his Department bulletin published and that if he wished to obtain priority of discovery it would be necessary to publish something on the subject very quickly. Whereupon he sent back the note which subsequently appeared in PHYTOPATHOLOGY (June, 1911).

Upon protesting to Dr. Giampietro after the appearance of his paper in the Rivista he said he had only sent a carbon copy of the English manuscript to the editor of that journal, without request for publication, and signed the following explanation :

"*Corrigendum.*—Through an oversight I omitted to mention in my article on bacterial onion rot (Rivista di Patologia Vegetale, July, 1911), that John R. Johnston, of this laboratory, had already shown *Bacillus coli* to be the cause of the bud-rot of the coconut palm, and that I was familiar with his conclusions. (Dated) September 18, 1911, Laboratory of Plant Pathology, U. S. Department of Agriculture." (Signed) A. W. Giampietro."—ERWIN F. SMITH.

The Cronartium associated with Peridermium filamentosum Peck. Through the kindness of Forest Assistant Frank B. Notestein of the Forest Service, who has supplied abundant specimens of *Peridermium filamentosum* for inoculation experiments, the leaves of two species of Castilleja, one of which had grown in the greenhouse, were inoculated with the aeciospores of this species of Peridermium, June 29, 1912. In about ten days, July 9, the uredinia of a species of Cronartium were noted on the leaves of both species, while control plants remained free

from infection. This confirms my prediction that a Cronartium on Castilleja is associated with *Peridermium filamentosum*. (See Mycolgia **4**: 142, May, 1912).

It is proposed that the name of this Cronartium be **Cronartium filamentosum** (Peck) comb. nov., as Dr. E. P. Meinecke reports proofs by inoculation that *Peridermium stalactiforme* Arthur & Kern is the aecial form of *Cronartium coleosporoides* (Dietel & Holway) Arthur.—GEORGE GRANT HEDGCOCK.

Dr. E. W. Olive, lately botanist at the South Dakota State College and Experiment Station, has been appointed curator of the Brooklyn Botanic Garden. Dr. Charles Brooks, botanist at the New Hampshire State College and Experiment Station, is appointed pathologist in the Office of Fruit Disease Investigations, Bureau of Plant Industry, and will begin work in his new position September 1. G. F. Gravatt has been placed in immediate charge of the work on the chestnut bark disease in Virginia; his address is Blacksburg, Va. J. B. Demaree, recently instructor in botany and plant pathology in the Maryland Agricultural College, becomes assistant botanist of the Ohio Experiment Station, in charge of diseases of truck and forage crops. H. W. Anderson, recently assistant in "Pathological Floriculture" at the Illinois Experiment Station, has been elected Professor of Botany in Wabash College.

According to the Experiment Station Record, a committee has been formed to solicit funds for the erection of a monument to the memory of A. Millardet, formerly Professor of Botany at the University of Bordeaux. Professor Millardet was the first to recognize the value of copper sulphate in combating the downy mildew and his investigations resulted in the formulation of Bordeaux mixture, one of the most efficient and widely used fungicides known. The treasurer of the committee is M. Andre Rozier, Gironde Agricultural Society, Bordeaux, France.

REVIEWS

Methods of Control of Plant Diseases in North America[1]

Paper read before the Royal Danish Agricultural Society, March 22, 1911.

Dr. F. Kœlpin Ravn, Professor of Plant Pathology in the Royal Agricultural High School of Copenhagen, Denmark, and the foremost exponent of the Rostrup school, in a well written paper gives an interesting report of observations and notes taken during a visit to the United States and Canada in 1910. His itinerary included, besides a prolonged visit to Washington, D. C., a great number of the more important Agricultural Experiment Stations and Colleges as well as a number of representative private institutions. By far the greater part of the paper is devoted to a rather detailed report on the latest results in American phytopathology and on problems under investigation during his visit, presenting an interesting resumé of the actual state of work in our country seen with the keen eye of an unbiased but sympathetic and well-trained foreigner.

What strikes Dr. Ravn most forcibly is quite evidently the combination of experimental scientific work in phytopathology with business principles and the computation of the cost of control work as against the actual saving in crop in dollars and cents, a feature somewhat neglected in Europe. With genuine appreciation he speaks of the development of scientific and practical work in the U. S. Department of Agriculture and its immense educational influence through cooperation with the various State Experiment Stations, through temporary field stations in a given focus of infection and through liberal distribution of literature. In studying the activities of the Department Dr. Ravn encountered the same difficulties as other European visitors on account of its organization ; he does not understand why the study of plant diseases in the broader sense of the word should be scattered over a number of different offices and even bureaus. He contrasts the remarkable cooperation of the Department with the farmer with the lack of cooperation between the various bureaus of the Department itself. As an example Dr. Ravn explains the existing difference of opinion between soil chemists and biologists as to the causes of "wheat-sickness" by the "unfortunate organization" both in Wash-

[1] Foranstaltninger til bekaempelse af plantesygdomme i Nordamerika. Foredrag, holdt i det Kgl. Danske Landhusholdningsselskab den 22. Marts 1911. af F. Kœlpin Ravn. Saertryk af Tidsskrift for Landbrugets Planteavl, 18. bind. 1911.

ington and in the different states which places all soil investigations in separate divisions, working altogether independently from the biological divisions.

In the Agricultural Experiment Stations throughout the country he is strongly impressed by the same close cooperation of the scientific staff with the practical farmer. According to Dr. Ravn this intensive cooperation, which is so characteristic of American experimental work has resulted in introducing methods for the control of plant diseases into all branches of agriculture to a degree unknown in most other countries with exception perhaps of the case of viticulture in Europe. This is particularly true of intensive cultivation of orchard trees, berries and vegetables, while the extensive cultivation of the commoner agricultural plants is far behind in this respect. The secret of success lies in the fact that the work of the American Experiment Stations has given practical results of greatest economical value. The Experiment Stations are therefore highly popular and have no difficulties in securing necessary funds.

The weakest point, in his opinion, is legislation with regard to control of plant diseases. The almost complete lack of federal legislation has placed all regulation of disease control in the hands of the individual states with resulting chaotic conditions throughout the country. He notes particularly that the only control against importation of plant diseases from foreign countries is found in California. On the other side Dr. Ravn was much interested to find in certain states (California and Oregon) the organization of cooperative control of plant diseases by all property owners of a certain locality regulated by law and placed in the hands of State commissioners with police authority. The discussion following Dr. Ravn's paper brought out the surprise felt by his Danish audience that such farsighted enforcement of control work should be possible in the "land of liberty."—EMILE P. MEINECKE.

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